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THE BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST

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NOTICE TO CONTRIBUTORS

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BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST

Vol. XLVIII. JANUARY, 1954 No. 1.

EDITORIAL

IN another two years this Journal will celebrate its golden jubilee. Since its foundation it has continued as an independent journal, interested in the social and preventive as well as the clinical aspects of disease, and it is, in fact, the oldest organ dealing with tuberculosis, diseases of the chest and cognate subjects. Its ever-widening range over the historical, preventive, industrial and clinical spheres is reflected in the large number of contributions which are being received from this country, the Dominions, the United States and Europe.

The policy of the Journal for 1954 involves certain changes, including a change of cover, a further increase in size of the Journal and inevitably an enlargement of its Editorial Board.

Our readers will recall that in January 1952 four distinguished Dominion colleagues from Australia, Canada, New Zealand and South Africa were welcomed. We have now decided to invite further members from the United Kingdom to widen representation in the provinces, and it is a pleasure to welcome Mr. Ronald Belsey (Bristol), Dr. Reginald Ellis (Manchester), Dr. A. G. Ogilvie (Newcastle-on-Tyne) and Dr. Maxwell Telling (Leeds). Their names and their contributions to the development of our knowledge of various aspects of tuberculosis and diseases of the chest are sufficiently well known to explain the benefits which will accrue from having these new members on our Board.

We much regret that during this process of growth we shall be losing the official services of an " old stalwart," Dr. Clifford Hoyle, who has promoted the interests of the Journal for sixteen years, thirteen as Editor and three as a valued member of the Editorial Board. Our sorrow at his resignation is tempered by the assurance that his experience will still be available to us unofficially. His replacement by a friend and colleague, Dr. Maurice Davidson, as a London representative will commend itself to our readers as an index of the high standard we are pursuing. Apart from his reputation in the clinical world, his experience in the realm of medical literature will be invaluable to the Editorial Board.

That the Journal is being read with interest and appreciation in the United States is shown by the appearance of an enthusiastic review of our 1952 volume in the October 1953 issue of *Diseases of the Chest*. Our readers will be gratified to learn that its Editor-in-Chief, Dr. Jay Arthur Myers, should write: " Each of

the four 1952 issues was outstanding in its content. . . . With this splendid array of editorial board members known everywhere for the excellence of their work and contributions in the field of diseases of the chest including tuberculosis there can be no doubt about the continued high quality of this Journal. . . . Current British workers are contributing so much to our knowledge in this field that one may expect a large number of excellent articles to appear regularly in British publications, including the BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST. Thus, each physician regardless of residence in the world should avail himself of the opportunity to read every issue of this Journal."

A NEW APPROACH TO THE PATHOGENESIS OF EXTRAPULMONARY TUBERCULOSIS*

By Hugh E. Burke

From the Royal Edward Laurentian Hospital, Montreal, Quebec, Canada

Certain recent studies have shown that some extrapulmonary lesions of tuberculous etiology, long held to be blood-borne in origin, are really the result of lymphogenous dissemination of tubercle bacilli. In 1940 the writer set out experimental, anatomical and clinical findings which confirm and amplify Souligoux's and Kaufman's observations concerning the mode of origin of spontaneous cold abscesses of the chest wall. In this paper the writer pointed out that some of his findings indicate that Kaufman's suggestion that tuberculous lymph nodes give rise to paravertebral abscesses, and even to disease of the spine itself, deserves serious consideration. Studies reported by the present writer (1947 and 1950) in recent years seem to leave little doubt that: (1) Spontaneous cold abscesses of the chest wall are the result of the transport of tubercle bacilli from the pleural space to the parasternal lymph nodes and the subsequent breakdown of tuberculous foci in these nodes; and (2) tuberculous paravertebral abscesses and tuberculous lesions of the spine are frequently the result of transport of tubercle bacilli from the pleural space to the para-aortic lymph nodes and the development and subsequent breakdown of caseous foci in these nodes.

During the experimental, anatomical and clinical studies just cited it became evident that the parasternal and para-aortic lymph nodes can, and probably should, be regarded as the regional lymph nodes of the pleural spaces.

Further reflection, re-examination of the work sheets describing the earliest experiments carried out prior to publication of the first of the above-cited studies, and scrutiny of the clinical findings in a number of patients with tuberculous extrapulmonary disease who have been seen in the past twelve years, have brought to light certain seemingly new and important observations concerning the pathogenesis of extrapulmonary lesions of tuberculous etiology other than spontaneous cold abscesses of the chest wall, paravertebral abscesses and tuberculosis of the spine.

The reflections to which reference has just been made are briefly as follows: One of the prime functions of the pleuræ is to reduce friction as the lungs increase and decrease in size during respiration. It is for this purpose that they have smooth surfaces bathed in secretions. Simple deduction suggests that the amount of secretion bathing the pleuræ varies from time to time, depending, among other things, on the rate and the depth of respiration. No one appears to have made a special study of the probable variations in the amounts of

* Read in part at the annual meeting of the Canadian Tuberculosis Association, held in Regina, Saskatchewan, on June 19 and 20, 1952.

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secretion bathing the pleuræ or more particularly of the means by which normally produced excess secretion is removed.

A. Experimental Findings

In the light of the foregoing reflections re-examination of the findings in the guinea-pigs which were given small intrapleural injections of colloidal thorium dioxide and/or finely divided lampblack, suggests that, at least in part, amounts of pleural secretion, in excess of immediate requirements, may be transported along lymph channels to the parasternal and para-aortic lymph nodes. Certain experiments which have been undertaken in recent weeks seem to lend weight to this view and, in addition, to indicate that the rate and/or the depth of respiration have a bearing on the speed with which, and the extent to which, normal secretions and particulate matter "escape" to the parasternal and para-aortic lymph nodes.

Perusal of the work sheets describing the experiments carried out prior to publication of the first of the series of studies cited in the opening paragraph of the present communication brought to light one experiment which was not reported at the time. This experiment is briefly as follows:

Ten Mantoux-negative guinea-pigs were injected intrapleurally with a small quantity of virulent human tubercle bacilli (Strain H_{37}) in suspension in isotonic saline. All of these guinea-pigs died within three weeks of the date of the intrapleural injection. All were autopsied. Presently available notes indicate that none of these animals had pleural effusions, that a few presented signs of localised pleural disease—small scattered tubercles on the visceral or the parietal pleura and/or some delicate pleuritic adhesions, that some had enlarged parasternal and para-aortic lymph nodes, and that all had miliary tubercles in the lungs, the liver, the spleen and the kidneys.

Two experiments similar to the one just described have been carried out in recent months. In one of these experiments ten Mantoux-negative guinea-pigs were injected intrapleurally with one-half a lethal dose of human tubercle bacilli (Middlebrook and Yegian, 1946). All of these animals died within ten to thirty days. Necropsy examination disclosed that all had tubercles on the pleuræ and/or a few pleuritic adhesions and, in addition, tubercles in the liver. In the second experiment a similar number of Mantoux-negative guinea-pigs were injected intrapleurally with one-fifth a lethal dose of tubercle bacilli (Strain $H_{37}Rv$). All of these animals died within five weeks to three months. Necropsy examination of these guinea-pigs revealed findings similar to those in the group given intrapleural injections of one-half a lethal dose, except that the pleuritic adhesions in these animals seemed to be a little more numerous and perhaps somewhat larger and, in some of those which lived more than six weeks, the peritoneum was studded with tiny tubercles. No evidence of pleural effusion was found in any of the animals in either of these two groups. The parasternal and para-aortic lymph nodes of these guinea-pigs did not appear to be enlarged. No macroscopic tubercles were noted in the lungs, spleen or kidneys of any of these animals. The spleens of all, however, were unusually large. (These experiments—the ones in which the effects of the rate and/or the depth of respiration on the "escape" of particulate matter

from the pleural space were studied and the ones just presented in abbreviated form—will be described in detail in other communications.)

These experiments, the previously unreported one in which Mantoux-negative guinea-pigs were employed and the ones which have been carried through in recent months, seem clearly to indicate: (1) That tubercle bacilli introduced into the pleural spaces of healthy guinea-pigs commonly evoke only a limited pleural response; and (2) that bacilli so introduced may be transported from these spaces to the parasternal and para-aortic lymph nodes, may " escape " from these nodes to the blood stream and so may become the means of causing tuberculous lesions to develop in various tissues and organs.

B. Observations on Clinical Material

In previously reported studies the writer has drawn attention to the fact that pleurisy, either of proved or presumed tuberculous etiology, is frequently a precursor of spontaneous cold abscesses of the chest wall, of paravertebral abscesses and/or of Pott's disease.

The clinical findings in a goodly number of patients with a great variety of extrapulmonary lesions of proved tuberculous etiology, seen over the past twelve years, reveal that many had had symptoms or signs of pleuritic disease, presumably of tuberculous origin, prior to recognition of their extrapulmonary lesions. Detailed examination of the records of these patients discloses that many had at least one thing in common—namely, that they took more exercise during the acute phase of their pleuritic disease than is generally considered desirable for persons known to have, or suspected of having, pleuritis of tuberculous etiology.

The case records of five patients with extrapulmonary lesions of known tuberculous origin, which typify some cases of non-pulmonary tuberculosis seen by the writer in recent years, are given below.

A.M., a 17-year-old girl, had had pain in her left chest and had felt tired at times for over a year prior to seeking medical advice in February, 1944. She then stated that to her knowledge her temperature had been elevated afternoons for several weeks. Examinations disclosed a left-sided pleural effusion. A sample of fluid removed for examination was clear and serous. No micro-organisms of the *M. tuberculosis* variety were found in this specimen. A Mantoux test (1-10,000 O.T.) gave a positive response. She was advised to rest in bed at home pending admission to a sanatorium. She soon began to have troublesome headaches and arrangements were made to admit her to a general hospital. She became very drowsy soon after admission. Examinations showed that she had tuberculous meningitis. *M. tuberculosis* was identified in a sample of her cerebrospinal fluid. She died in coma on March 5, 1944.

W.S. was 55 years of age when he came under the writer's supervision. Inquiry revealed the following: Patient was exposed to gas in November 1917, in France. He seems to have suffered no ill-effects from the same. All X-rays taken in the three following years were reported to show no evidence of pulmonary or pleural disease. In 1936 the patient was seized with sharp pain in one side of the chest and a few days later with similar pain in the other side. He was advised to have first one side of the chest, then the other,

strapped and to give up work and rest at home. The patient remained away from work for a few days when the pain was severe, then returned to work. He had a further slight attack of pleurisy, on the left side, in 1937. The following year he applied for a pension because he had a cough and his chest was sore. A chest X-ray taken at this time was reported to show evidence of residual pleuritis on the right side. Request for pension was denied. Patient reports that he felt out of sorts between 1942 and 1944, when he had a further attack of dry pleurisy on the right side. Then in late 1945 he came down with a " cold in the prostate " and sought medical advice. He was told that he had tuberculous epididymitis and orchitis and was operated on in May 1946. Epididymis and testis removed at this time contained tuberculous disease. The right kidney was found to be the site of a tuberculous process the following year and was removed. A year later, the left testis began giving trouble. It was removed in July 1948. Examination showed that it was the site of tuberculous disease. This operation wound failed to heal in spite of streptomycin and para-amino-salciylic acid therapy. The patient first came under the writer's observation in 1952. He had no complaints at this time other than that his wound would not heal. A chest X-ray picture taken at this time disclosed signs of a right-sided residual pleuritis.

M.L., a young man of 21 years who was first seen by the writer in early 1951, was taken ill with a so-called " virus " pneumonia in December 1949. He began to have pain in his left chest soon after he came under observation. He was admitted to a general hospital the latter part of January (1950). Examinations disclosed a clear serous effusion in his left pleural space. This fluid proved to be negative for *M. tuberculosis* both on culture and guinea-pig inoculation. His Mantoux test (1-10,000 O.T.) was positive. He was discharged to take the cure at home, pending admission to a sanatorium. He took very little rest at home principally, as he says, " because he felt well." When he was admitted to a sanatorium in late March nearly all roentgenographic evidence of his pleural effusion had disappeared. He was given in consequence only limited bed rest. Shortly after entering the sanatorium he noticed a swelling on the dorsal surface of his left wrist and a pinhead-size lump on his left thumb. The nature of these abnormalities was not determined. He spent four months in the sanatorium. When given his discharge he was told that he might start light work in the autumn. In January 1951 the lump on his left thumb became painful. A dressing containing a balsam compound was applied. The lump broke down. He sought medical advice and was admitted to a general hospital for investigation. Examination showed that he had a tuberculous tenosynovitis of the left wrist, a tuberculous ulcer on his left thumb, minimal pulmonary tuberculous and residual pleuritis, presumably of tuberculous etiology. *M. tuberculosis* was identified in cultures of purulent material taken from the ulcer on his left thumb.

H.D. came under the writer's observation approximately one and a half years ago, when he was 25 years of age. He reported that he began to be " out of sorts " in early 1946; he often felt tired, had no interest in food and as a result lost weight. He became really ill at Easter, when he suddenly began to cough and to have severe pain in the left half of his chest; he sought medical advice at this point. He was found to have an acute pleurisy with effusion on the left side and was, in consequence, put to bed at home pending admission to a sanatorium, which he entered in early August. Examinations made at this time disclosed signs of a small encapsulated effusion and some thickening of the pleuræ, especially at the base, on the left side. He was

given only ordinary bed rest during his stay in the sanatorium and was discharged after four months. His diagnosis at the time of his discharge was: Pleurisy with effusion, presumably of tuberculous etiology—improved. He entered university the autumn of 1947, led an active life, and as far as he knows was well until early 1949, when he had a fleeting attack of low back pain. In February 1950 he began to have severe pain in his right buttock, sought medical advice, was put to bed for two weeks and obtained some relief, but experienced return of pain as soon as he began to move about again. In August (1950) his medical adviser recommended admission to hospital for investigation. Examinations disclosed signs suggestive of a ruptured intervertebral disc between L.5 and S.1. An exploratory operation was performed. This disclosed a tuberculous process involving the bodies of L.5 and S.1 and the adjoining tissues. Acid-fast bacilli, which later proved to be of the *M. tuberculosis* variety, were found in pus lying close to the vertebræ just described. A few weeks after this operation a spinal fusion (L.3 to S.2) was carried out and the patient was put in a hip spica cast. While in a cast and on bed rest, signs of a mass appeared in the right lower quadrant of his abdomen. This mass proved to be a psoas abscess. Pus aspirated caused generalised tuberculosis in guinea-pigs. Patient was admitted to, and discharged from, hospital a number of times between early 1951 and early 1952. In spite of treatment, his right-sided psoas abscess failed to resolve and a left-sided psoas abscess appeared and began to discharge externally. Acid-fast bacilli were found in smears made from pus obtained from the latter abscess. Routine examinations made when patient was first seen in consultation in February 1952 failed to disclose any evidence of pulmonary or pleural disease, revealed signs of a seemingly satisfactory spinal fusion (L.3 to S.2) and disclosed a bulging right-sided psoas abscess and, as noted, a draining left-sided psoas abscess. Special examinations revealed signs of an abscess overlying the lower lumbar and upper sacral vertebræ. A perforated polyethylene tube was introduced into this abscess by way of the draining left-sided psoas abscess. This, in conjunction with appropriate chemotherapy, resulted in prompt disappearance of the bulging right-sided psoas abscess, gradual cessation of drainage and seemingly complete healing of the left-sided psoas abscess.

C.G., 24 years of age when she came under the writer's observation on November 23, 1951, reported that she had had pleurisy two and a half years previously. Inquiry revealed that she had had typical pleuritic pain on the left side; that her temperature, which was elevated when she entered hospital, returned to a normal level in a matter of two days, and that she was allowed to leave hospital, seemingly without specific instructions as to what she should do, approximately four weeks after falling ill. As far as she knows she was well from the time that she left hospital until June of the year that she came under observation. In the early summer of 1951 she began to have bouts of weakness and dizziness accompanied by fever. These often lasted for two or three days and were sometimes accompanied by pain of a pleuritic type in the left half of the chest. She noticed that she often felt tired at the end of the day and, by midsummer, became aware of the fact that she was losing weight. In early August she began to cough and to raise sputum. She sought medical advice at this time and was told that she had a pleurisy with effusion and advised to enter a sanatorium. Examinations made at this time disclosed signs of active moderately advanced pulmonary tuberculosis. *M. tuberculosis* was found in a specimen of sputum collected on August 21. Her cough all

but disappeared soon after she entered the sanatorium. She continued, however, to complain of pain in the left half of her chest and sometimes in the opposite side. Her temperature fluctuated between 100° and 101°F. On September 30, while receiving chemotherapy and being kept on strict bed rest, she began to complain of severe pain in the lower portion of the right half of the chest and in the right shoulder, and her temperature rose to 104°. Examinations made at this time disclosed signs of a right-sided pleural effusion. Her temperature remained at high levels throughout October and November, in spite of many and varied forms of therapy. Numerous attempts to discover the reason, or reasons, for her failure to respond to therapy yielded nothing of note until mid-November, when a roentgenogram of the spine disclosed signs of narrowing of the disc between the bodies of D.10 and D.11, and possibly some narrowing of the disc between D.9 and D.10. Patient came under the writer's observation at this time. She was obviously ill. Her temperature was 99·4° F. It remained elevated, often going to 105·8° for weeks thereafter. Special examinations carried out in the period immediately following her transfer to this hospital's Montreal treatment unit confirmed the presence of active moderately advanced pulmonary tuberculosis, of a right-sided pleural effusion, and of Pott's disease involving three of the lower dorsal vertebræ. These examinations also disclosed the presence of a large paravertebral abscess lying alongside the vertebræ just described. On December 13 a needle was introduced into the paravertebral abscess. About 20 c.c. of purulent material was removed. Examination of this material disclosed many acid-fast bacilli. Following this procedure the patient's temperature fell to approximately a normal level. She improved to such an extent between January and June that in October it seemed advisable to undertake a spinal fusion and simultaneously open drainage of her paravertebral abscess. Her progress since operation has been satisfactory.

Discussion

While the observations just recorded were being collected and assembled, Roper and Waring (1952) read an unusually interesting and informative paper, before the American Trudeau Society, on a follow-up study of 141 cases of sero-fibrinous pleural effusion. Roper and Waring's cases were men in whom sero-fibrinous pleural effusions developed while they were on active military duty in World War II. All had had routine chest roentgenograms interpreted as "negative" within approximately one year of the onset of pleuritis. Each case exhibited a positive Mantoux test during the course of his effusion. All were observed frequently and periodically for five or more years after the onset of their original effusion or until death. Six of the 141 persons included in this study died within three years of the onset of their original effusion—five from tuberculosis and one from carcinoma of the larynx in addition to active pulmonary tuberculosis. Of the 141 patients, 92 (65 per cent.) became ill with tuberculosis nine months or more, usually within three years, after the onset of their initial pleural effusion. Practically all forms of tuberculosis were represented in the 92 patients whose disease relapsed. Pulmonary tuberculosis without recurrence of effusion developed in 57 persons; pulmonary tuberculosis with recurrence of effusion, in 18 cases. In 16 of the 92 men reactivation took the form of recurrent pleural effusion without manifest pulmonary disease. In one person extrapulmonary disease,

seemingly without pulmonary or recurrent pleural involvement, developed. In addition to this one case of "pure" extrapulmonary tuberculosis, eight of the men in whom pulmonary tuberculosis without recurrence of effusion appeared, four of those who relapsed with a combination of pulmonary disease and recurrence of effusion, and six of those whose reactivation took the form of a recurrence of effusion seemingly without pulmonary disease, had extrathoracic extensions of their tuberculous process. These nineteen persons with extrathoracic tuberculosis had twenty-three instances of involvement—namely, ten involvement of lymph nodes, one of an eye, one of the larynx, three of bones and joints, two of the gastro-intestinal tract, two of the genito-urinary tract, two with a perirectal abscess, one with miliary disease, and one with meningitis. All five of the men who died from tuberculosis had extrathoracic disease of tuberculous etiology. The extrathoracic lesions in these men became manifest soon after they had seemingly recovered from their original pleural effusion. After making detailed analyses of a number of factors which might have a bearing on the probability of relapse, Roper and Waring came to the following conclusions: (1) That the bacteriological findings in, and the maximum volume of the initial pleural effusion; the degree of toxæmia, and the laboratory findings, during the initial illness; and the status of the post-effusion chest roentgenograms, do not appear to bear any relationship to the probability of relapse; but (2) that the younger the patient and the shorter the length of time that the patient is required to rest in bed for the original illness with effusion, the higher the relapse rate is likely to be. As evidence for the relationship between age and relapse, they state that 55 (75·3 per cent.) of the 92 who relapsed were between 18 and 24 and the remainder—37 (54·4 per cent.)—were between 25 and 45 years of age. They report that in their study of the relationship of the length of bed rest to relapse they felt obliged to set aside thirteen patients because they developed manifest tuberculosis while they were still in hospital and as a result were given periods of bed rest longer than would otherwise have been the case. They state that 66 of the remaining 128 persons underwent bed rest for periods up to six months; that 60 (90·9 per cent.) of these relapsed; that 62 rested for six months or more; and that only 19 (30·6 per cent.) of this latter group relapsed.

Viewed in the light of previously reported studies and of the experimental and clinical observations recorded in the present communication, Roper and Waring's contribution seems to indicate that under certain circumstances tubercle bacilli may be transported from the pleural space to remote parts of the body and thereby become the means of inciting extrapulmonary lesions of tuberculous etiology. In previous studies it was reported that tubercle bacilli introduced into the pleural spaces of Mantoux-positive guinea-pigs commonly evoke a striking pleural response and concomitant changes in the parasternal and para-aortic lymph nodes. All of the animals included in these studies were killed at intervals during the six weeks following the intrapleural injection of tubercle bacilli. When tubercle bacilli are introduced in the pleural space of Mantoux-negative guinea-pigs the course of events is quite different from that in Mantoux-positive guinea-pigs. Necropsy examination of these guinea-pigs regularly disclosed signs of minimal pleural response, in a few instances enlarged parasternal and para-aortic lymph nodes, in some

tubercles in the lungs, spleen and kidneys, and in all tubercles in the liver. These guinea-pigs regularly died within a few weeks of the date of the intra-pleural injection—those injected with one-half of a lethal dose, in less than thirty days, and those injected with one-fifth of a lethal dose, within five to twelve weeks.

Certain experiments to which passing reference is made in this communication seem to indicate that the rate and/or the depth of respiration affect the speed with which, and the extent to which, particulate matter—namely, finely divided lampblack—" escapes " from the pleural spaces to the para-sternal and para-aortic lymph nodes. Obviously it is impossible in both the small group of persons with extrapulmonary tuberculosis cited in the present paper, and in Roper and Waring's series of 141 young men whose initial manifestation of tuberculosis was a sero-fibrinous pleural effusion, to make any statement with respect to the number of tubercle bacilli which incited the initial pleural response or the relationship between the time of arrival of these micro-organisms in the pleural space and the advent of tuberculin sensitivity. Available evidence suggests that all of the patients in whom pleurisy with effusion was the initial evidence of tuberculosis were Mantoux-positive prior to the time that tubercle bacilli entered the pleural space. In spite of the lack of information with respect to the numbers of tubercle bacilli concerned and the tuberculin sensitivity of the persons involved prior to recognition of their pleuritic disease, it seems probable, in the light of the observations reported herein, that in some of the patients whose findings are recorded in the body of this paper and in some of Roper and Waring's patients tubercle bacilli were transported from the pleural space to the parasternal and/or the para-aortic lymph nodes and then, from these nodes, were carried into the blood stream and on to distant tissues and organs. The fact that certain experimental studies suggest that the rate and/or the depth of respiration bear a direct relationship to the speed with which, and the extent to which, particulate matter is transported from the pleural spaces to their regional lymph nodes also seems to lend weight to the view expressed in the body of this paper, and confirmed by Roper and Waring's observations, that if patients with proved or suspected tuberculous pleuritis are not given every encouragement to go to bed following the onset of their disease and to remain there for several months, they are prone to fall ill with tuberculosis, either in the form of pulmonary disease or more particularly extrapulmonary disease, within a few years of the onset of their pleuritis.

Summary

Certain observations which seem clearly to indicate that most spontaneous cold abscesses of the chest wall and many instances of Pott's disease are due to the transport of tubercle bacilli from the pleural spaces to the parasternal and the para-aortic lymph nodes respectively, and the subsequent breakdown of caseous foci in these nodes, are reviewed.

Evidence is presented which suggests that the parasternal and para-aortic lymph nodes may, and probably should, be regarded as the regional lymph nodes of the pleural spaces. Reference is made to certain experiments which

seem to indicate that the rate and/or the depth of respiration influence the speed with which, and the extent to which, particulate matter is transported from the pleural spaces to their regional lymph nodes.

Certain experiments in which Mantoux negative guinea-pigs were injected intrapleurally with various doses of virulent tubercle bacilli are described. These experiments show that guinea-pigs injected intrapleurally with supposedly sublethal doses of tubercle bacilli commonly die within a few weeks from widely disseminated tuberculosis. Necropsy examination of these animals revealed pleural changes of a minor nature (a few tubercles and/or some delicate adhesions) and tubercles in the liver of all, and enlarged parasternal and para-aortic lymph nodes and tubercles in the lungs, spleen and kidneys of some.

The findings in five subjects who typify persons with extrapulmonary tuberculosis seen by the present writer in recent years are presented in some detail. These five patients have at least one thing in common—namely, that they took more exercise during the acute phase of their pleuritic disease than is generally considered desirable for those known to have, or suspected of having, pleuritis of tuberculous etiology.

The pathogenesis of extrapulmonary tuberculosis is discussed in the light of the foregoing and of a recent paper by Roper and Waring describing the results of a follow-up study of a series of cases of sero-fibrinous pleural effusion.

The findings presented herein seem clearly to indicate: (1) that some forms of extrapulmonary tuberculosis—namely, spontaneous cold abscesses of the chest wall and in some instances paravertebral abscesses and Pott's disease—are due to the transport of tubercle bacilli from the pleural spaces to the parasternal and the para-aortic lymph nodes respectively, with subsequent breakdown of caseous foci in these nodes and extension of the tuberculous process to adjoining tissues; and (2) that certain other forms of extrapulmonary tuberculosis—for example, genito-urinary tuberculosis—may at times be due to the transport of tubercle bacilli from the pleural spaces to the nodes just described with subsequent " escape," at least of some of these micro-organisms, into the blood stream and lodgment in distant tissues or organs.

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TUBERCULOUS PERICARDITIS

By H. John Anderson

From St. Thomas's Hospital, London

Introduction. Tuberculous infection of the pericardium presents in its evolution special problems of diagnosis and treatment and, in addition, produces circulatory changes which lead to a characteristic clinical picture. The advent of antibiotics may well present an increased number of patients who have survived acute tuberculous pericarditis but who continue to suffer from residual pericardial disease in the form of a scar. These considerations make the problem of pericardial tuberculosis one of particular interest to the physician and surgeon.

Terminology. Manifest tuberculosis of the pericardium, if it evolves fully, passes from effusion to fibrosis and healing, and at any time the action of the heart may be embarrassed. For clinical purposes I propose to make an arbitrary division of this continuous process into three stages—acute, subacute and chronic—and when the action of the heart is embarrassed I shall call the result restrictive pericarditis. My reason for using this term so generally is to emphasise the essential identity of the circulatory changes in all phases. I prefer the descriptive term "restrictive" to "constrictive," because I believe it more accurately describes the process.

Incidence. An incidence of 0·7 per cent. in 1,000 autopsies was found by Osler (1893), 1·1 per cent. in 7,219 autopsies by Norriss (1902), and Suzman (1942) found 0·4 per cent. in 1,500 autopsies. These figures relate to anatomically proven tuberculosis of the pericardium, and fail to record those cases where the process has healed completely or left a non-specific fibrous scar.

The records of White (1935), of Peel (1948) and of Pickering (1948) all include such cases in which proven tuberculosis of the pericardium has healed rapidly to a non-specific fibrohyaline scar. Three of my cases have shown non-specific fibrosis in the resected pericardium following recovery of the tubercle bacillus from a pericardial effusion; and I think it likely that the real incidence of tuberculosis of the pericardium is higher than autopsy evidence suggests.

Material. This paper is based on an experience of 26 patients with proved pericardial tuberculosis and 12 patients with polyserositis which progressed to restriction. It is convenient to group these in the following way:

Group I: Where pericarditis was an incident in the course of disseminated tuberculosis: 8 cases.

Group II: Where proved tuberculous pericardial effusion settled with apparently complete recovery: 5 cases.

Group III: Where tuberculous effusion was present at the outset and the disease progressed to chronic restriction of heart action: 8 cases.

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Group IV: Where chronic restrictive pericarditis was present at the outset and tuberculous pericarditis was found to be the cause: 5 cases.

Group V: Where the disorder began as a polyserositis and progressed to chronic restriction of the heart action but no proof was obtained of a tuberculous basis of the disease: 12 cases.

Mode of Infection. Direct hæmatogenous involvement of the pericardium is occasionally found in patients dead of disseminated tuberculosis, but is clearly not the common mode of involvement, because miliary tuberculosis is not a common finding early in the disease even although it may occur later.

Retrograde spread from tuberculous tracheobronchial lymph nodes involved in primary tuberculosis was suggested by Scott and Beattie (1938) as a result of their observations of infection in primates; and in twelve of my cases a primary tuberculous lesion of the lung and lymph nodes was demonstrated either by X-rays or after death.

Rarely, direct extension into the pericardium may occur from a tuberculous gland, lung or chest wall, and very rarely a softened gland may rupture into the pericardial sac. One case of particular interest in the evolution of the histological picture showed a histologically tuberculous gland inseparably attached to a restricting pericardial scar of non-specific histology.

Morbid Anatomy. The specific changes of tuberculous infection of any serous membrane are too well known to need repetition.

There are, however, certain points of interest to the clinician. Tuberculous infection of the pericardium may become arrested at any stage. It may be merely fibrinous (Peel, 1949) or show a large or small effusion which is absorbed or replaced by caseous or granulation tissue, which may restrict heart action for weeks or permanently, or may resolve at length and leave a normal heart.

In the process of healing caseous abscesses may long remain walled off within masses of fibrous pericardium, and at operation such an abscess may be opened and cause tuberculous involvement of the pleura; or such an abscess may involve the myocardium—in one case it extended through the ventricular wall to the endocardium. Spread from such an abscess may be responsible for a sudden late progression of restriction of the heart action.

The visceral and parietal layers of the pericardium are often densely thickened and yet separated from each other by looser tissue, as is shown in the specimen illustrated (Fig. 1). The inner layer alone produces a material degree of restricted heart action, and herein lies the explanation of the fact that, although the initial aspirations of a tuberculous effusion may give great circulatory relief, the later aspirations are often ineffective. It is also essential for the surgeon who deals with the problem to remove the inner layer from the myocardium: a troublesome and dangerous manœuvre.

The pericardium over the left ventricle posteriorly is often the portion most involved, as the illustration shows (Fig. 2); this is probably because the effusion collects here first and in greater quantity (Connor, 1925). It is essential for the surgeon to remove this portion of the pericardium and this should be done first, for, if the right ventricle is first relieved of restriction, acute pulmonary œdema may develop.

Tuberculous pericardial effusions are often blood-stained and even frankly bloody. Polymorphs may be found in the early stages, but lymphocytes later

predominate. Tubercle bacilli are almost never seen in direct smears and they are difficult to recover even by guinea-pig inoculation or by culture. When the pericardium is removed several portions should be examined microscopically.

Many records show that minor degrees of liver fibrosis are found in cardiac failure of long duration, and gross degree of cirrhosis may be found in those cases of tuberculous pericarditis which progress to chronic restriction of the heart.

Increased hepatic venous pressure, decreased hepatic arterial flow, decreased appetite due to gastric congestion with nutritional hepatic cellular insufficiency, are all factors in producing this fibrosis, the significant results of which are ascites, diminished plasma proteins and occasionally diminished prothrombin formation. These result in increased surgical hazard and occasionally in ascites which persists after the pericardium has been excised.

A more or less gross thickening of the liver capsule is sometimes found (Zuchergussleber) and, while the origin of this may rarely be tuberculous infection, it is far more likely that this fibrous proliferation is due to the greatly increased lymph flow in the subcapsular lymphatic spaces. This occurs when the hepatic venous pressure is raised, as was shown by Bolton and Barnard (1920) and Wolweiller (1950).

The myocardium is abnormal in acute and chronic pericarditis and these factors contribute to the abnormality.

The coronary arteries are encased in fibrous tissue and this may adversely affect the dynamics of coronary flow. Certainly it makes difficult the task of the surgeon who strips the pericardium, and in one case a coronary vessel was damaged and this caused an extensive myocardial infarction.

Roberts and Beck (1941) have shown in man and by experiment a 30 per cent. reduction in size of individual heart muscle fibres, which also become more uniform in diameter and circular instead of oval in cross-section.

This atrophy may be due to several causes; disuse; diminished blood flow, and possibly nutritional deficiency which results from congestive hepatic cirrhosis; repeated paracentesis; isolation of proteins in serous effusions, and reduced protein intake due to ill-health and impaired appetite caused by gastric congestion.

Subepicardial myocarditis has been stressed by Wolf as a cause of cardiac insufficiency.

In long-standing restrictive pericarditis there is often a layer of fat underlying the fibrous envelope over the left ventricle (Barnard, W. G.) and this can only have originated by fatty alteration of muscle fibres previously in this situation.

Focal myocardial change is often found and may be due to ischæmia caused by tuberculous involvement of blood vessels.

Active tuberculosis and also fibrosis in the auricular wall, especially the left (Heimann and Binder, 1940), is not rare and explains the intimate blending of auricle and pericardium which the surgeon has learned to respect, and also explains abnormalities of auricular rhythm. Rarely, a tuberculous abscess is found in the muscle of the ventricle, and one of my patients had two such abscesses.

Of 26 patients who had tuberculous pericarditis, 16 had pleural effusions,

10 bilateral, 3 right-sided and 3 left-sided, and in only 2 of those was the pleurisy proved to be tuberculous.

The problem is to differentiate effusions due to tuberculous pleurisy from effusions which result from the circulatory changes. There is no certain means of doing this. The protein content of the fluid is of little value. The experimental work of Volweiller (1950) and his colleagues has shown that ascites produced by constriction of the inferior vena cava and the hepatic veins has an average protein content of 2·5 grammes per cent., and may have as much as 4·4 grammes per cent.

Conversely constriction of the inferior vena cava and portal vein causes ascites with a protein content of 0·3 gramme per cent. These observations reinforce the conclusion that non-inflammatory transudates may have greatly varying protein content—0·3 to 4·4 grammes per cent.

The cytology of exudates and transudates is similar and also gives little assistance. Culture and guinea-pig inoculation are seldom positive even when the serous membrane is known to be tuberculous.

Occasionally the pleura reacts to tuberculous infection with the production of a dense layer of fibrous tissue of considerable thickness, and produces a frozen hemithorax.

I have seen this five times in restrictive pericarditis as is illustrated (Fig. 3) here; the pericardium and pleura are similarly involved, the thickness of the pleura is over half an inch. There was no histological evidence of tuberculosis, although tubercle bacilli had been found in the pericardium eighteen months before.

This condition may occur primarily, or follow operation (Burwell and Ayer, 1941) across the left pleura when the pericardial disease is still active, and it may then add a serious additional burden to the patient. But it may be that chemotherapy and the use of antibiotics will prevent this complication.

Rate of Formation of Effusion and Cardiac Output. In acute restriction of the heart the most important single factor is the rate of formation of the effusion, because the rise in intrapericardial pressure depends on the relative inelasticity of the fibrous pericardium. In rapid accumulations, such as occur from hæmorrhage into the pericardium, this inelasticity quickly limits increase in size of the sac, and further rapid increase takes place by compressing the cardiac chambers and extinguishing the circulation.

When the fluid accumulates less rapidly the fibrous parietal pericardium is gradually stretched and may reach an enormous size and contain 2 or more litres.

As the intrapericardial pressure rises so the pressure within the heart in diastole rises; and restricted filling of the heart in diastole results in decreased output per beat. If the heart rate remains unchanged the cardiac output per minute is correspondingly reduced, but in most cases some degree of tachycardia at rest partly compensates for the fall in stroke output; and it is only by extraordinary tachycardia that the increased demands of exercise are met.

All observers are agreed that the venous pressure is invariably raised in restrictive pericarditis, but the existence of venous pulsation in restrictive pericarditis is still not widely recognised—as lately as 1949 references are made to "apparent venous pulsation." The veins do pulsate in restrictive pericarditis, and at times it may even be that the venous pulsations are so vigorous

that they are mistaken for arterial pulses. Often the pressure is so high that the upper level is above the neck in an upright position, and it may even be above the forehead, when pulsation is difficult to detect.

Five patients had obvious liver pulsation, and the form of venous pulse suggests that the cause is not tricuspid incompetence but an unusually large difference between the systolic and diastolic venous pressure.

Mechanical types of obstruction (Fig. 4). When chronic pericarditis restricts the heart the mechanical results of this change are not identical in every case. As already illustrated the bulk of the abnormal material is usually over the left ventricle (1). This prevents diastolic filling of the left ventricle by preventing relaxation; or sometimes, by encroaching on the mitral valve (2) in the auriculo-ventricular groove, prevents filling of the left ventricle as in mitral stenosis; or, very rarely, the pulmonary veins are stenosed as they pass through the pericardium to enter the left auricle (3).

The last mechanical state causes the symptoms of pulmonary congestion, especially orthopnœa, with radiographic evidence of pulmonary congestion, and in the electrocardiogram prominent P waves and right ventricular preponderance, or a gradually increasing right preponderance.

This predominant left-sided restriction is not common in uncomplicated cases, though it was not very rare when surgeons used the anterior approach to the pericardium—which allowed the right ventricle to be released more easily than the left ventricle. That it was not even commoner was due to the fact that few patients survived a sudden release of the right ventricle which produced immediate overloading of the lungs and massive pulmonary œdema. This happened in two of my patients who died and to one who had gross post-operative pulmonary œdema but survived.

At times the major restriction has affected the right ventricle (4) and the right auricle has remained relatively free. This type of mechanical obstruction behaves like tricuspid stenosis, and leads to excessive auricular and peripheral venous pulsation and liver pulsation.

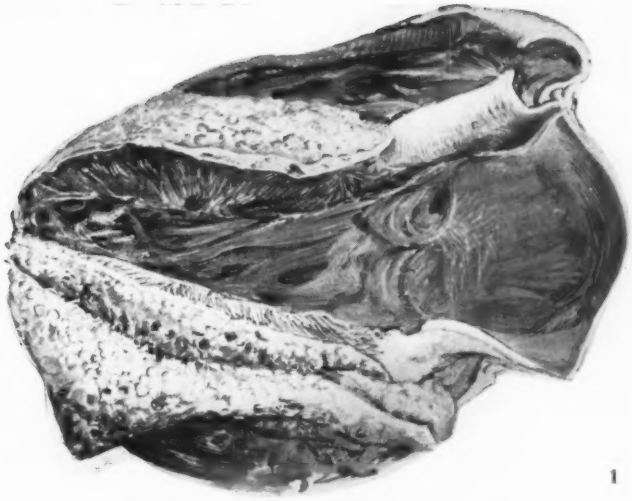
Very uncommonly the entry of the great veins through the pericardium is narrowed (5), but while it is sometimes suggested that failure to improve after pericardial resection is due to vena caval stenosis, anatomical studies do not support this view. This condition should not be difficult to distinguish by cardiac catheterisation which would show high superior or inferior vena caval pressure giving place to normal pressure when the obstruction is passed (White *et al.*, 1948).

Diagnosis. In diagnosis the disease falls naturally into three stages:

(1) *Fibrinous Pericarditis.* The symptoms are usually non-specific and relate merely to the general disease.

Occasionally in fibrinous pericarditis pain may be felt in the shoulder or in the root of the neck, and the neck may be tender to touch. At other times the pain is due to involvement of the pleura and is then felt to one or other side of the sternum and is related to respiration. Many patients feel an ill-defined ache vaguely localised somewhere beneath the sternum. Occasionally the type of pain suggests that it may be caused by myocardial ischæmia, perhaps due to coronary artery spasm or intra-pericardial pressure preventing an adequate coronary circulation.

PLATE I



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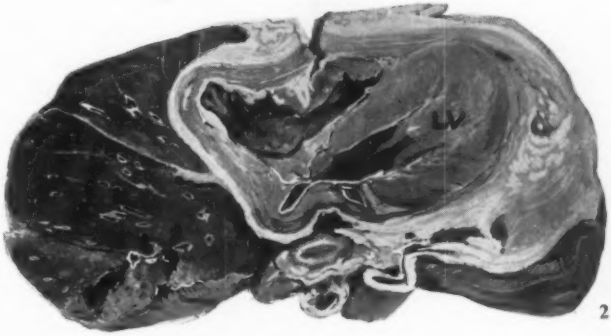
. 1.—Note thickening of parietal and visceral ericardium by tuberculous anulation tissue, with a ell-defined plane of cleavage between.



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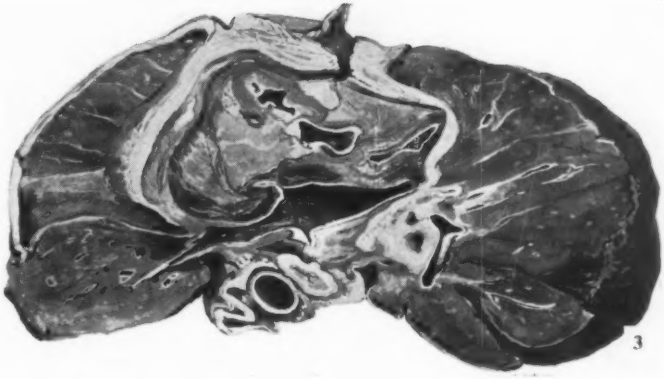
. 2.—Showing the greater ickness of the pericardium steriorly, over the left ntricle, in chronic tuberculous pericarditis.



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. 3.—Showing involvent of the left pleura by he tuberculous process.

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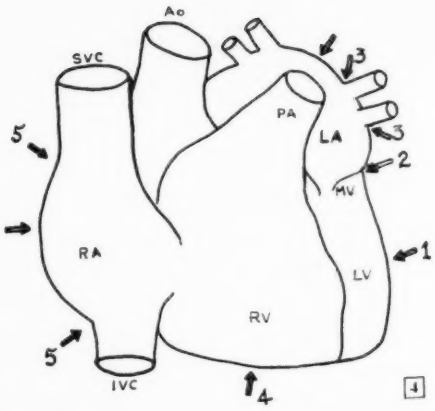

FIG. 4.—To illustrate varieties of obstruction due to pericardial fibrosis and thickening. 1, Mainly left ventricular obstruction. 2, Obstruction of the mitral valve in the atrio-ventricular groove. 3, Pulmonary vein obstruction. 4, Mainly right ventricular obstruction. 5, Obstruction to superior and inferior vena cava.

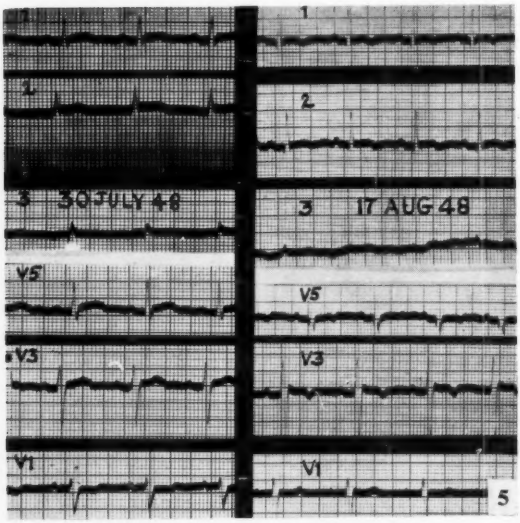

FIG. 5.—Electrocardiogram showing transi ST segment elevation in the acute stage pericarditis, with subsequent T-wave version.

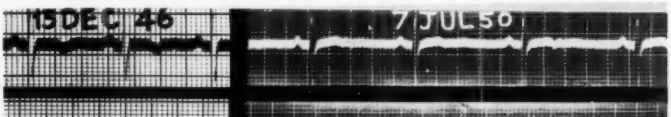

FIG. 6.—Showing cha in the electrocardiogram verting towards norma

Pericardial friction may be heard anywhere over the heart, but most constantly over the xiphisternum with the patient sitting up with the chest in full expiration.

Immediate discovery of the cause of fibrinous pericarditis is often impossible and the diagnosis is established by waiting. No doubt some patients with tuberculous pericarditis are seen in this stage and the process then heals.

Differentiation from rheumatic pericarditis is usually easy, but it is interesting that three patients gave a history of typical migratory arthritis and this may cause confusion. Pericarditis secondary to specific and virus pneumonias, or due to invasion of the pericardium by bronchial neoplasms or the reticuloses, or associated with myocardial infarction, can usually be differentiated.

The electrocardiogram is particulary useful in diagnosis of the fibrinous stage. There is often an elevation of the S-T segment—especially in lead 2, with preservation of the normal upward concavity of the S-T segment (Porte and Pardee, 1929). It is also seen in the chest leads—and is distinct from the changes of myocardial infarction in being maximal in lead 2 and in the form of the S-T segment (Fig. 5). This elevation may be transient or persist for a week or longer.

As the fibrinous stage proceeds to effusion the QRS complexes become smaller, the S-T segment becomes flat and the T waves inverted (Peel, 1934) (Fig. 5).

(2) *Serous Pericarditis.* Tuberculous effusion into the pericardial sac is commonly associated with few symptoms. A patient may walk into hospital with a huge effusion, which is causing only minor symptoms, little fever, and no great tachycardia.

On the other hand, dyspnœa and orthopnœa are sometimes extreme, and an adult will sit upright and lean forward to get comfort while a child may find greatest ease by lying face downwards. A small dry cough, often repeated and very troublesome, is usual, and is probably caused by pressure on the bronchi by the enlarged paricardium or enlarged lymph nodes. Chest pain is unusual.

To this form of pericardial disease particular diagnostic attention has been directed. The physical signs relate either to the increased size of the pericardium or to the circulatory changes which are caused by the effusion.

The cardiac dullness is increased, friction is often heard even when the effusion is massive, because anteriorly the heart and pericardium remain in contact (Connor, 1926). In almost all cases the signs first described by Ewart (1896) and still often ascribed to Bamberger are found—these are impaired percussion note with intense bronchial breathing and ægophony just below the angle of the left scapula. These signs are due to collapse of the left lung caused by the pressure of the pericardium, or to an associated pneumonic consolidation (Levin and Gevalt, 1940), or to a left pleural effusion (Gordon, 1940); but most commonly to the first of these, because there may be no pleural effusion and the signs may persist for weeks. These signs are responsible for some errors in diagnosis, for they may suggest a left basal pneumonia. With a very large pericardial effusion these signs may be bilateral.

The circulatory changes are a raised venous pressure, a low arterial pressure, pulsus paradoxus and peripheral circulatory stasis.

X-ray films of the heart show the typical pear shape, with loss of the normal details of contour; and in lateral films the posterior inferior recess shows a convex bulge due to collection of liquid. In addition the hilar vascular shadows are truncated by the heart border, and there is little or no vascular congestion in the lungs.

When the patient is screened the same points are seen in addition to diminished pulsations.

The electrocardiogram may show either S-T segment elevation such as already described, or small QRS complexes with flat S-T segments and flat or inverted T waves.

To establish with certainty the tuberculous origin of a pericardial effusion is difficult unless there is obvious primary or miliary tuberculosis of the lungs or chronic pulmonary tuberculosis, though the last is uncommon.

The character of the effusion is not helpful; it is often blood-stained and tubercle bacilli, in my experience, have not been found in direct smears. Culture on artificial media and guinea-pig inoculation are not always positive. Out of nine histologically proven cases where a guinea-pig was inoculated, five were positive and four were negative.

Effusion into the pericardium has to be distinguished from cardiac enlargement due to any cause—particularly diffuse myocarditis. The shape of the heart is similar, pulsations may be deficient in both, and the electrocardiogram may be similar. A final decision may be made on the history of the illness and the electrocardiogram, which in diffuse myocarditis may show a prolonged QT interval (Tung), whereas the QT interval is normal in uncomplicated pericardial effusion. Even then differentiation may be impossible and it may be necessary to needle the pericardium.

From rheumatic pericardial effusion differentiation is usually easy on the history, but I have already pointed out the occurrence of arthritic pains in tuberculosis and two of my patients had initially been treated as rheumatic pericarditis. Wood makes a point of interest and diagnostic value; he finds that half his cases of proven rheumatic pericarditis have none of the expected electrocardiographic changes and suggests that diffuse rheumatic myocarditis overshadows the subepicardial myocarditis, on which the characteristic S-T segment changes depend.

Effusion secondary to malignant invasion is usually blood-stained, and this may cause diagnostic confusion for a time; cytological examination of the effusion for malignant cells by Dudgeon's wet method may settle the diagnosis and should always be done.

Pyogenic pericarditis is associated with considerable general upset and leucocytosis.

Less easy to differentiate at the onset are cases of so-called benign pericarditis, such as Loque and Windkos (1948) describe, where the effusion is lymphocytic and complete recovery takes place within a few weeks.

(3) *Fibrocaseous and Calcareous Pericarditis.* As the pericardial effusion absorbs, the heart becomes smaller and is surrounded, at first by a layer of fibrocaseous material and later by a fibrous sheet, the thickness of which varies in different cases: this is responsible for the variation in heart size. Less than 10 per cent. are greatly enlarged and most are within normal limits.

An interesting aspect of patients with chronic restriction is the fact that they may develop heart failure quite suddenly; one developed it with a chill and another while running to catch a train; as if acute myocardial failure had suddenly been added to chronic mechanical embarrassment.

These patients are often well, although there is active tuberculosis of the pericardium, and a failure to find the symptoms and signs of general disease should not count against the diagnosis.

The symptoms are effort dyspnœa, abdominal swelling, œdema of the feet and legs, and in children and young adults œdema of the face and arms. Occasionally the main complaint is coldness and blueness of the extremities and, rarely, fainting with exercise.

Where the restriction of the heart is unbalanced, so that the left ventricle is more completely restricted, orthopnœa and attacks of pulmonary œdema may be found, but this is unlike the usual patient who can lie recumbent in comfort.

The venous pressure is invariably raised and the liver always enlarged and firm, sometimes pulsating. Most patients have some œdema of the ankles. The heart rate is increased at rest and slight exercise increases it abnormally with a slow return to the resting rate. Pulsus paradoxus is common but the really obvious degrees of this abnormality are found while the effusion is developing. The systolic blood pressure is low, usually between 100 and 110 mm. of mercury, with a small pulse pressure; but in four patients it was 150/80, 150/100, 140/100, 140/100 and 140/90 mm. of mercury.

The force of cardiac pulsation is diminished, the heart sounds are quiet, a mid-diastolic third heart sound is common, but a disastolic shock and systolic retraction are rare.

X-ray may show calcification in the pericardium, screening shows minimal pulsation, and where the pericardium is thick the barium swallow is deviated in the right oblique position and simulates an enlarged left auricle. Peel (1948) stresses this and says a radiological diagnosis of mitral stenosis may be made.

Two-thirds of the patients with restricted heart action have abnormally small QRS complexes and inverted T waves in the electrocardiogram.

Harvey and Whitehill suggest (1937) that the form of the electrocardiogram should be watched, for where the T waves revert towards normal the prognosis is good for spontaneous recovery from restriction.

After excision of the pericardium the electrocardiographic changes revert toward normal (Fig. 6).

White (1948) has pointed out that in some cases where operation has failed to cure the patient a change in the electrocardiogram to right axis deviation with large P waves suggests the left ventricle has been inadequately freed, or that there is mechanical obstruction in the left atrio-ventricular groove simulating mitral stenosis.

Myocardial failure without great increase in heart size is not common, it is found when the right ventricle fails as a result of excessive load; but as most examples of chronic cor pulmonale are secondary to respiratory disease, the presence of pulmonary abnormality makes these obvious. However, primary pulmonary hypertension is difficult to diagnose, and it is essential to make the distinction because patients with pulmonary hypertension are bad subjects for surgery.

Hepatic cirrhosis should offer no real difficulty in diagnosis because engorgement of the jugular veins does not occur.

Nevertheless, many patients with restrictive pericarditis are in fact diagnosed and treated for months and years as though suffering from primary hepatic fibrosis.

If the condition of the jugular veins were always noted, this mistake would become obsolete. Furthermore, the electrocardiogram is normal in cirrhosis.

Rarely a massive thrombus within the auricle or an intra-auricular new growth, such as a pseudomyxoma, has to be differentiated. From the literature these cases would appear in the past to have defied diagnosis. My own experience is of a single case of auricular pseudomyxoma, where the clinical picture was that of restrictive pericarditis. Exploration showed a normal pericardium, and at post-mortem the true cause was found. Today intracardiac catheterisation and angiocardiography should assist.

Very rare causes of heart disease, such as primary systemic amyloidosis and the so-called idiopathic myocardial failure, have been mistaken for restrictive pericarditis.

Certain points require stress. Pulsating jugular veins and a pulsating liver do not exclude restrictive pericarditis; the systolic blood pressure is not always low; the third heart sound in mid-diastole may cause confusion with mitral stenosis; auricular fibrillation is not rare, especially in patients aged over 30 years.

Treatment. Patients who have tuberculosis of the pericardium require treatment for two conditions—tuberculous infection and circulatory embarrassment.

The infection should be treated with streptomycin in single doses of 1 gramme daily by intramuscular injection, and Paraminosalicylic acid 5 grammes by mouth four times daily. I suggest a total dose of at least 100 grammes of streptomycin.

Where it is necessary to aspirate the pericardium 2-5 grammes of streptomycin can safely be introduced at each aspiration.

The use of streptomycin has improved the outlook where the tubercle bacilli are found in the effusion, probably because it controls miliary dissemination.

It is too early to say with certainty whether the local disease heals more rapidly and completely with streptomycin, but three patients had pericardial effusions in which bacilli were found, and all have recovered completely within twelve months.

The treatment of the circulatory changes falls into three phases.

The first, in which acute restriction is caused by the quantity and pressure of effusion within the pericardium. This should be treated by paracentesis if there is restlessness, much dyspnœa, orthopnœa or peripheral cyanosis, and when pulsus paradoxus is obvious or the systolic blood pressure is low or falling.

The pericardial sac is easily explored from several points, an approach through the left costo-xiphisternal angle is safest, but we should be prepared to use more than one approach because the effusion may become loculated.

In the literature it is clear that pericardial aspiration is often considered to be a last resort, and not a few patients have suffered unnecessarily and some have certainly died because it was not done.

Enough fluid should be removed to alleviate the symptoms and improve the circulation, and this will frequently be 5 or more ounces; but it is notable that successive aspirations of the pericardium have less effect in improving the circulation, because the visceral layer when thick may alone cause restriction.

The introduction of air into the pericardium after aspiration of effusion has been suggested as a means of preventing adhesions between visceral and parietal layers. I am doubtful of its efficacy, and whether adhesion between these layers is necessarily bad, or whether the state of the visceral layer, which may alone cause restriction, is affected by air replacement.

In the second phase of the circulatory disorder the effusion is replaced by caseous material and the disease is still active.

Healing of the lesion begins at its inception, and the problem is to know when the disease is no longer active in the sense that restriction will occur again after operation.

Homans says that removal of the pericardium in the acute stage is safe and his cases have done well; with this I agree. Four of my cases of caseous tuberculous pericarditis have recovered following operation. Some cases recover, however, without operation, and unless restriction is severe, or increases rapidly, a period of six to twelve months' treatment with antibiotics and chemotherapy should be tried before operation, because there is, during this period of time, a real possibility that spontaneous recovery will occur, as it did in five of my cases, even after a year's restriction.

There is no great risk that congestive hepatic cirrhosis will cause permanent portal hypertension with ascites during this waiting period, for there are many patients recorded who have had severe restriction for years and have nevertheless made a complete recovery after operation.

However, where restriction is severe enough to endanger life—and this is rare—the pericardium should be removed; the operative risk is small and miliary dissemination should not occur as a result of operation if antibiotics are used.

While waiting for the disease to heal and the circulatory changes to disappear, pleural effusions which cause dyspnœa, and ascites which causes discomfort, should be removed; fluid and sodium intake should be restricted and mercurial diuretics given.

A diet rich in protein should be given to maintain the plasma protein level against internal plasmaphoresis and protein loss from paracentesis.

The use of digitalis is controversial.

In some cases it has been demonstrated that the cardiac output is reduced by digitalis (Burwell and Straghorn, 1932; Lyons and Burwell, 1946), and it is therefore thought to be useful only when auricular fibrillation complicates restrictive pericarditis, or when the heart dilates following pericardial resection.

While this experimental work is convincing there are patients who are undoubtedly improved by digitalis, and this is probably because they have some myocardial failure in addition to mechanical restriction.

As there is no means of measuring the separate effects of these factors, digitalis should be given, and a careful assessment made of its effect by observation of urine output and weight.

If it proves ineffectual it can be stopped. To condemn its use absolutely may be to fail to use a drug which greatly assists management of the disease.

In the third phase the circulatory disorder is caused by a fibrous scar or a calcified shell in which there may be no evidence of tuberculosis.

How long it is before this stage is reached is not certain, but where the scar is fibrous it will usually be at least a year after the onset, although in one case an entirely non-specific scar was found eight months after recovery of tubercle bacilli from the effusion.

Once it is decided that restriction is not likely to disappear under conservative treatment the pericardium should be resected.

To wait longer is probably to risk increased myocardial atrophy, more extensive congestive cirrhosis, further depletion of plasma protein, and in addition to prolong invalidism.

When it is decided that an operation is necessary, it should be possible to assess the exact site of the mechanical restriction, by correlating the symptoms, physical signs, electrocardiogram, and the results of cardiac catheterisation with intracardiac pressure readings. With this knowledge the surgical attack on the pericardium can be launched accurately and the circulatory embarrassment relieved.

Certain points relative to operation are important. Adhesions between the pericardium and surrounding structures are of no importance (Parkinson, 1930; Hosler and Williams, 1936; Armstrong, 1940). The left ventricle should be freed from restriction before the right ventricle. The anterior approach to the pericardium is unsatisfactory because it is more likely to leave the back of the heart restricted and to result in acute or chronic pulmonary œdema. The innermost layer of the pericardium must be removed down to heart muscle because even a thin fibrous layer remaining is sufficient to cause restriction.

The removal of pleural effusions and ascites before operation increases vital capacity, but the latter has also the effect of leaving the stretched abdominal wall unsupported and makes coughing difficult. The abdominal wall should, therefore, be supported by an abdominal binder after paracentesis abdomini.

Blood loss is particularly dangerous, because it causes the venous pressure to fail and further reduces cardiac output.

After excision of the scar return to normal is sometimes disappointingly slow, but recovery even after months of such inertia is not uncommon and it is not possible to predict the rate of recovery. Failure to recover may be due to one or more of several causes. The innermost layer of the pericardium may not have been removed; at other times the left ventricle has been incompletely freed and this results in chronic pulmonary congestion; or the surgeon may have injured the blood supply of the heart; or the disease may be still active and envelop the heart again.

Occasionally tuberculous pleurisy or peritonitis may cause persistent effusions and falsely suggest an incomplete relief of the restrictive process. Where the venous pressure returns to normal after operation it will be clear that the effusions are not due to restriction. All cases are not so simple, however. One patient had persistent ascites and a right pleural effusion after operation, and also a raised venous pressure; it was thought the effusions were due to restriction. A second operation showed pleural tuberculosis.

Most often, however, recovery is slow for no well-defined reason, and it is probable that myocardial changes previously described have been added to the mechanical effects of the pericardial and take time to recover.

Summary

Tuberculous pericarditis is not a rare disorder; it has, in common with other forms of chronic pericarditis, particular effects on the circulation which result in morbid anatomical changes in various organs. The diagnosis and management of the acute, subacute and chronic stages of the process are discussed.

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CHRONIC DESCENDING PULMONARY TUBERCULOSIS

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The term chronic pulmonary tuberculosis includes any variety of that disease which runs a prolonged course, and therefore covers many types of disease which differ in radiographic appearance and in pathology. This paper describes one type which can be distinguished radiologically, which remains symptomless for many years, causing problems in diagnosis and management, and which responds dramatically to antibacterial treatment, thus stressing the need for early diagnosis and treatment.

The ten cases which form the present series were encountered personally during four years at the Brompton Hospital between 1949 and 1952. Selection depended upon satisfying the radiographic criteria which are the only means of identifying the early stages of the disease. Its frequency in comparison with other types of tuberculosis could not be assessed, but its comparative rarity is undisputed.

Radiographic Features. Cases were identified on the following characteristics:

1. Pinhead opacities equally distributed in the supraclavicular areas of both lungs.
2. Slow bilateral symmetrical development of fresh opacities immediately below those seen in previous radiographs.
3. Slow but continuous descending spread of opacities to involve all zones of both lungs symmetrically, without cavitation or coarse infiltration.

In the earliest radiographs opacities were discrete, well defined, 1-3 mm. in diameter, and were first seen in the supraclavicular areas of both lungs. At this stage they are extremely difficult to identify even in good-quality full-sized radiographs, and are unlikely to be recognised in miniature radiographs. Progressive changes which followed were the essential criterion for recognition, but they were slight and insidious and could not be detected by comparing radiographs taken three months apart. To be certain it was necessary to compare radiographs taken one or two years apart, when fresh opacities were recognised immediately below those seen in the earlier film (Fig. 1). A series of radiographs covering many years (Figs. 3 and 4) demonstrated the slow bilateral symmetrical spread from the apex towards the diaphragm, giving the appearance of descending curtains of infiltration. All ten patients showed these serial changes and, although the rate of spread varied, it was always slow. The time taken to reach the lower zones depended on the extent of disease when first discovered. In seven patients disease spread until opacities involved the lower zones. In four the earliest radiograph showed opacities confined to the apices, and an average of seven years elapsed before the lower zones were

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involved. In the remaining three patients the earliest radiograph showed disease below as well as above the clavicles, and in these an average of three years elapsed before further spread involved the lower zones. As spread continued the older opacities became less distinct (Fig. 2), and when they reached the lower zones those in the upper zones were fused by linear shadows suggesting that fibrosis had occurred, causing gradual elevation of hilar shadows. Opacities were least concentrated in the mediastinal and diaphragmatic areas of the lungs, and in many patients tomography demonstrated that the opacities were evenly distributed within the lungs.

Although this paper is primarily concerned with the early recognition of cases during the prolonged first stage, subsequent changes and deterioration must be considered. All patients in the present series progressed without remission unless antibacterial treatment was given, and in five patients deterioration continued after descending opacities had involved both lower zones. Cavitation developed in these five patients, all cavities being thin-walled, under 4 cm. diameter, and situated in upper lobes or in apical segments of lower lobes. In three cavitation persisted and further changes were seen, consisting of rapidly spreading irregular opacities resembling those seen in bronchogenic spread of caseating tuberculosis (Fig. 4). Changes of both clinical and therapeutic significance occur when cavitation develops, and again when irregular opacities begin to spread rapidly. It is therefore convenient to regard the advent of cavitation as the second stage, and of spreading irregular opacities as the third stage in a slow but continuous process.

This descending spread of pinhead opacities is pathognomonic of this variety of chronic pulmonary tuberculosis and is not seen in any other disease. However, the first available radiograph may show that all zones are already involved, so that recognition of further descent is impossible, and serial radiographs may only reveal increased concentration of opacities in the lower zones, or subsequent cavitation and rapidly spreading irregular opacities. Thus the later the first available radiograph the greater the difficulty in recognising the progressive changes.

Clinical Features. Six patients were male and four were female and all were between twelve and twenty-five years of age at the time of the first abnormal radiograph (Table 1). Four patients whose first radiograph showed opacities confined to supraclavicular areas averaged fifteen years of age (range twelve to eighteen years). In the remaining six the first abnormal radiograph showed more extensive disease, and their average age was twenty-one years (range nineteen to twenty-four years). This age incidence suggests that the disease often begins during adolesence between the ages of twelve and eighteen. Seven had no previous chest disease, one had had a pleural effusion and two had had mild asthma which did not recur during the period of observation. Two patients were contacts of patients with pulmonary tuberculosis, but the remainder gave no history of contact with or familial susceptibility to that disease. Nine patients were English, one was Polish and none was Jewish; no patient was robust and five were below average physique. In nine there was no history of occupational risk of pulmonary disease; the tenth had been an electrician but had not been exposed to beryllium. The outstanding clinical feature in every case was the absence of symptoms for many years despite extensive and

TABLE I.—SUMMARISED FINDINGS IN TEN CASES OF CHRONIC DESCENDING PULMONARY TUBERCULOSIS.

Case Number	*Sex*	*Age in years at first abnormal radiograph*	*Appearances of earliest abnormal radiograph*	*Years elapsing before finding tubercle bacilli*	*Appearances of ultimate radiograph*	*Result of antibacterial treatment*	*Observed duration of improvement*
1	M	15	Supraclavicular opacities	4	Opacities all zones, and cavitation	Total clearing	3½ years.
2	M	15	Supraclavicular opacities	3	Opacities all zones, and cavitation	Total clearing	1½ years.
3	M	17	Upper zone opacities	2	Opacities all zones, cavitation, and spreading irregular opacities	Slight clearing of opacities	None.
4	M	19	Upper zone opacities	1	Opacities all zones, cavitation, and spreading irregular opacities	Extensive clearing of opacities all zones: cavities persisted	1½ years.
5	M	22	Upper zone opacities	2	Opacities upper and mid zones	Extensive clearing	1 year.
6	M	24	Upper zone opacities	1	Opacities all zones	Extensive clearing	½ year.
7	F	12	Supraclavicular opacities	None found	Opacities all zones	Not treated	—
8	F	20	Upper zone opacities	4	Opacities all zones	Extensive clearing	2½ years.
9	F	21	Upper zone opacities	4	Opacities all zones, cavitation and spreading irregular opacities	Extensive clearing all zones: cavities disappeared	2 years.
10	F	24	Upper zone opacities	2	Opacities upper and mid zones	Extensive clearing	2½ years.

progressive disease. Seven patients were symptomless when first radiographed; two were recovering from bronchitis and one from pleurisy. All were observed for between two and six years following the first abnormal radiograph. No patient developed significant lasting symptoms while the radiographic appearances were confined to descending pinhead opacities (stage one). Of five who deteriorated further and developed cavitation (stage two), all had a persistent cough, two had small hæmoptyses and two developed small pleural effusions. Three patients deteriorated still further and had irregular spreading opacities (stage three), and they then complained of toxæmic symptoms commonly experienced in severe tuberculosis. The early radiographic changes

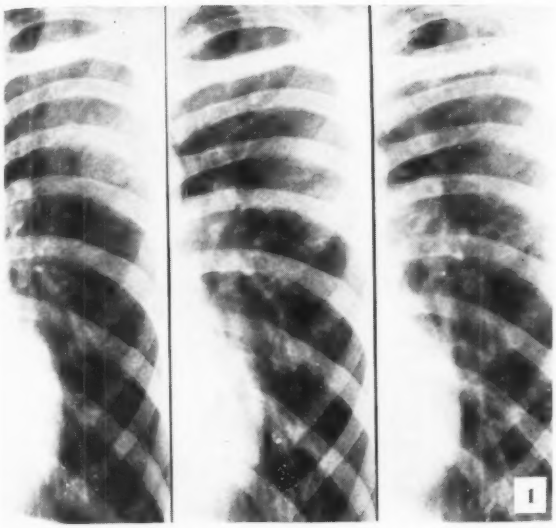

FIG. 1.—Radiographs (Case 1) taken at yearly intervals four years after spread began, illustrating the difficulty in recognising spread by comparison of radiographs taken at yearly intervals.

FIG. 2.—Radiographic appearance of upper zone opacities (Case 1) four years after spread began.

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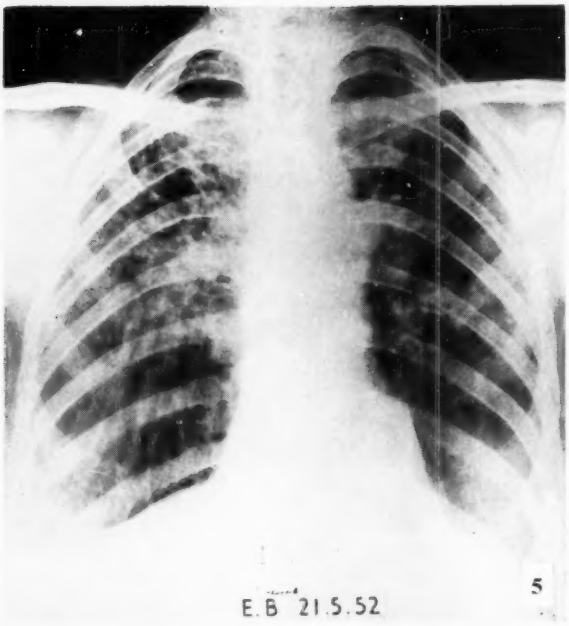

FIG. 5.—Chest radiograph of a female aged 33 years showing diffuse bilateral disease known to have been present eight years previously.

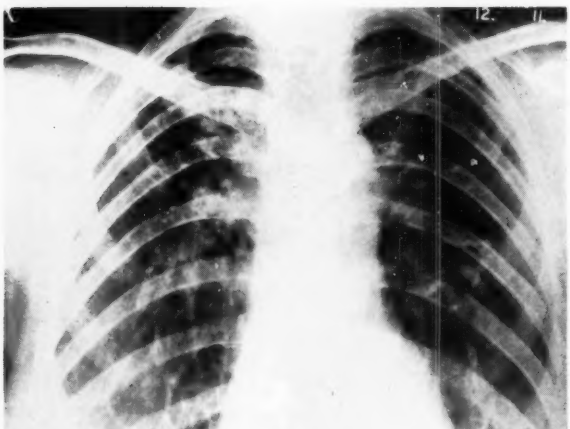

FIG. 6.—After six months' anti-bacterial treatment there was considerable clearing of opacities with persistence of linear shadows.

gave rise to no abnormal physical signs; and only in the advanced stages of the disease were apical râles heard. The erythrocyte sedimentation rate was normal in six out of eight patients examined within a year of discovery and it was only slightly raised in the remaining two.

Aetiology and Bacteriology. Tuberculin skin sensitivity tests were positive in each of nine patients tested, and tubercle bacilli were ultimately recovered from all of them by examination of sputum or gastric contents. In no patients were tubercle bacilli found when an abnormal radiograph was first discovered, however extensive the disease. In nine patients with disease and bacteriological

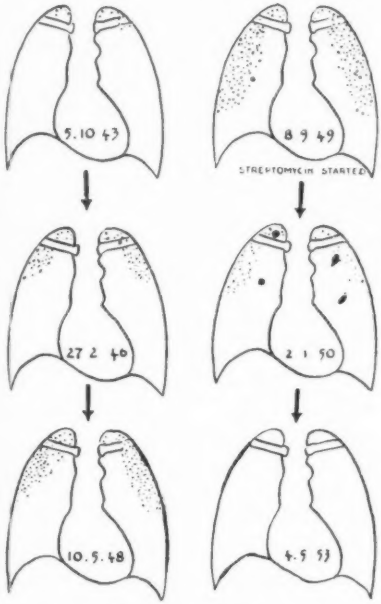

FIG. 3. — Diagrams illustrating radiographic appearances (Case 1), showing the descending spread of opacities during six years, with apical cavitation (8.9.49). After treatment cavities became opaque and opacities cleared in an ascending direction.

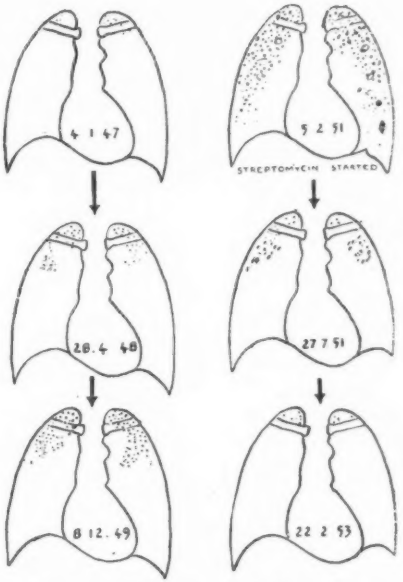

FIG. 4. — Diagrams of radiographic appearances (Case 9) showing descent of opacities into lower zone, cavitation and irregular infiltration (stage 3). Antibacterial treatment was followed by clearing of opacities in ascending direction and disappearance of cavities.

confirmation, an average of two and a half years elapsed before tubercle bacilli were first discovered, and in many instances this was an isolated finding not confirmed until many years later. This characteristic difficulty in obtaining bacteriological confirmation during the prolonged first stage cannot be exaggerated, although there was comparatively little difficulty in finding tubercle bacilli during the final stages.

Treatment. Eight patients observed before 1949 were treated by one or more periods of rest at home or in hospital and six also received sanatorium treatment. All eight patients subsequently resumed an active life and in none was there evidence that either prolonged rest or resumed activity had altered

the progress of the disease, which spread uninterruptedly unless antibacterial treatment was given. Pulmonary relaxation was attempted in five patients. Artificial pneumothorax was unsuccessful in two because no pleural space was found; two others were treated by pneumoperitoneum, combined in one instance with phrenic crush, the remaining patient receiving phrenic crush alone. In none of these patients was there evidence that relaxation combined with rest had interrupted the progress of the disease.

Nine patients received antibacterial treatment, all receiving daily streptomycin combined with P.A.S., but the duration of treatment varied. Eight patients who continued treatment for two months or longer and received more than 50 gm. of streptomycin all improved; the ninth failed to respond, but was known to have had cavitation and a persistently positive sputum for two and a half years before sustained antibacterial treatment was given. Radiographic improvement occurred in eight patients and was first seen in the lower zones with clearing of recently developed opacities (Figs. 3 and 4). Improvement was often suspected two or three months after treatment began, but changes were so slight that a further two months was generally required to be certain of this improvement. After clearing had begun symmetrically in the lower zones it continued in an ascending direction towards the apices, in the reverse direction to that in which opacities appeared. Clearing in the upper zones was often incomplete with persistence of linear shadows, but complete clearing was observed in two patients sixteen months after treatment began; their disease was confined to upper and middle zones, being discovered three and six years previously. Clearing was greatest in patients with limited disease of comparatively recent development, but considerable improvement was also observed in patients whose disease was known to have been present for over five years. Improvement was maintained throughout the period of observation in seven out of eight patients, the eighth relapsing after a severe illness eighteen months after treatment began. The fate of cavities varied with their type and length of history. In three patients thin-walled cavities disappeared and did not return, but apical ring shadows persisted in two others, in one of whom bronchography demonstrated cystic bronchiectasis. In two patients cavities became opaque during antibacterial treatment (Fig. 3), but later disappeared completely.

The benefits of early streptomycin treatment have encouraged attempts to recognise this disease in patients with an incomplete series of radiographs and sometimes on a single film. Since bacteriological proof is frequently unobtainable, the radiographic differentiation of diffuse nodular opacities can be difficult (Scadding, 1952), but careful examination of the nature and distribution of the individual foci is generally helpful, for in few other diseases are uniform small dense opacities of symmetrical apical distribution seen. Reference has already been made to the difficulty in identifying cases if the disease is extensive when first discovered. Among older patients radiographs have been seen which, although excluded from the present series, showed extensive disease similar in character and distribution to that observed in the present cases. Some of these patients showed radiographic improvement after antibacterial treatment, and opacities cleared in the same ascending direction suggesting further similarity with the present well-defined series. To illustrate this important group Fig. 5

shows the radiographic appearances of a lady aged 33 years, whose extensive bilateral disease was stated to have been present eight years previously. Her freedom from symptoms and the radiographic appearances suggested a similarity with the present series and the possibility that the extensive disease when first radiographed at the age of 25 had precluded recognition of the characteristic descending spread. When early radiographs later became available, careful comparison suggested an increased concentration of opacities in both lower zones during eight years, and after tubercle bacilli were isolated from her sputum antibacterial treatment was begun. After six months' treatment there was clearing of opacities in lower and mid zones (Fig. 6) in an ascending direction. It is therefore probable that some cases outside the strict definition of the present series may have a similar pathology and derive equal benefit from antibacterial treatment, which must therefore be considered when similar radiographic appearances are encountered.

Discussion

The cases here reported show a striking uniformity in respect of their clinical features, their radiographic appearances and their response to antibacterial therapy, and although pathological data are not available, interest is aroused in regard to continuous radiographic deterioration in the absence of clinical symptoms, and to the nature of the unusual descending spread of disease.

In some tuberculous patients freedom from symptoms may be explained by high resistance, by the limited quantity of disease seen radiographically, or by radiographic evidence of arrested disease. Other factors may be present which are believed to modify the clinical response to infection, such as the age of the patient, the presence of racial, familial or of constitutional resistance to tuberculosis, and the presence of extrapulmonary tuberculosis. In the present series of cases none of these factors was of significance. All patients belonged to an age group susceptible to severe tuberculosis, all were of slight physique and extrapulmonary tuberculosis was found in none. Therefore some further explanation should be sought to explain their widespread bilateral disease with radiographic evidence of continued deterioration lasting many years without causing symptoms.

Descending radiographic spread of any variety of pulmonary tuberculosis has been reported infrequently. Furthermore most of the examples reported have differed from the present cases in that opacities were neither uniform nor small, and that spread was both irregular and asymmetrical. Ornstein, Ulmar and Dittler (1931) mentioned chronic proliferative tuberculosis in their classification and described this as fine apical infiltration which "descended over the course of years." Reisner (1934) described an apico-caudate spread of disease in eight patients among 102 with pulmonary tuberculosis. Reporting the results of streptomycin treatment in chronic tuberculosis, Howlett and O'Connor (1948) gave diagrammatic illustrations of one patient with similar spread of fine infiltration, and Levine (1949) gave diagrams showing descending nodular disease in symptomless patients. Although pathological descriptions are included in some of these papers, in none is there information on the pathology of a case which specifically showed this descending spread of disease. Patho-

logical descriptions of other types of chronic pulmonary tuberculosis have been given by many authors, including Burnard and Sayé (1924), Pagel (1933), Pinner (1934), Sayé (1936), but in their patients the size and distribution of the foci differed from the present series and in no instance was the characteristic descending spread reported. When extrapulmonary tuberculosis was found to co-exist, many authors assumed that pulmonary foci resulted from hæmatogenous spread, although Pinner (1945) observed that this opinion was based on circumstantial evidence alone. Material for histological examination was not available in the present series. Patients do not die of the disease during the prolonged first stage, and they recovered when antibacterial treatment was given. If the disease progresses and patients die after developing cavitation and rapidly spreading irregular opacities, the pathology of early lesions will be obscured by the terminal fulminating tuberculosis. Autopsies of patients dying of this disease are therefore an unlikely source of information regarding the cause of this descending spread of opacities. Accidental death of a previously diagnosed patient, or the removal of adequate tissue by biopsy, might provide this information, but until accurate data are available the pathological basis of this unusual descending spread must remain speculative.

Tuberculosis of the lungs commonly spreads by aspiration through air passages and by hæmatogenous dissemination, and the resulting disease generally follows a recognisable radiographic distribution. The distribution of disease resulting from bronchogenic aspiration is generally asymmetrical and uneven, often being lobular or segmental. Such appearances were seen in none of the present patients. Disease resulting from hæmatogenous dissemination frequently involves both lungs equally, without sparing the lower zones as was characteristically seen in early stages of the present cases. Rubin (1939) drew attention to spread of disease via the bronchial arteries, but disease which resulted was generally uneven and asymmetrical. Although these radiographic appearances were never described, certain authors have suggested bronchogenic and hæmatogenous dissemination to explain descending spread of disease. Reisner (1934) disregarded the co-existence of extrapulmonary tuberculosis and suggested that the earliest pulmonary lesions were intimately related to the peripheral bronchioles into which they later ruptured and spread by intracanalicular progression. Levine (1949), believing that pulmonary spread was intermittent, suggested that it occurred by bronchiolar aspiration, delays occurring while bacilli gathered in numbers sufficient to permit aspiration into contiguous lobules. Present experience suggests that detailed examination of a complete radiographic series covering many years will show that deterioration is continuous although slow. Continuous symmetrical descending spread was observed in every one of the present cases and is difficult to explain on the basis of bronchogenic aspiration. Hæmatogenous pulmonary tuberculosis can be widespread yet symptomless. Hoyle and Vaizey (1937) have reviewed this subject, but only 13 out of 60 patients reported showed continued radiographic deterioration, and a symmetrical descending spread was described in none. The papers of Reisner (1934), Pinner (1934) and Wilkinson (1937) all mentioned descending spread of pulmonary disease in patients with extrapulmonary tuberculous foci. Similar foci were also present in the patients with chronic pulmonary tuberculosis described by Burnand and Sayé (1924), Davis and

Wilson (1938), Cohen (1940), Mann (1946) and Pagel (1948), but in these descending pulmonary disease was not reported. In most of their patients pulmonary disease was discovered as a result of X-raying patients known to have skeletal or genito-urinary tuberculosis, and in considering the cause of the pulmonary disease the understandably high incidence of extrapulmonary tuberculosis, presumably blood-borne, may have strengthened the assumption that pulmonary disease resulted from hæmatogenous dissemination. Lloyd Rusby (1952) reviewed these papers under the heading hæmatogenous tuberculosis but after examining the evidence in favour of spread via the blood stream he concluded that the assumption was based on analogy and was unproved.

If theoretical objections are raised against the routes by which spread frequently occurs, an alternative should be sought. Lymphatic spread of pulmonary tuberculosis is well recognised and examples have been reported by Schürmann (1929 and 1930), Hoyle and Vaizey (1937) and others, but the radiographic appearances of their patients were distinctive and consisted of linear striations radiating from the hila. Clegg (1953) has drawn attention to tuberculosis of endobronchial lymphatics which, although a common finding in overt tuberculosis, may also occur in bronchi which are normal to bronchoscopic and to naked-eye examination, and in parts of the lungs which show no radiographic abnormality. Disease can spread by endomural lymphatics, and in the present cases it is possible that early lesions lying in relation to the periphery of the bronchial tree may slowly extend by this path. Rapid extension would be resisted by lymphatic tissue at bronchiolar bifurcations. Minimal symptoms might be expected in tuberculous disease confined, as in primary infections, to pulmonary lymphatics, whereas rapid spread might occur if disease spread outside this system. In the present cases rapid deterioration only occurred after many years of progressive disease and after cavitation had developed. Tuberculosis confined for many years to the pulmonary lymphatics may account for the unusual clinical features seen in the present cases, and bilateral apical disease slowly spreading to contiguous endobronchiolar lymphatics may be the basis of the descending spread of disease seen in radiographs. Histological information is still required in these interesting patients, and it is hoped that by wider recognition a full pathological interpretation will become available in the future.

Conclusions

The dramatic and lasting improvement which followed antibacterial treatment of the present patients after other methods of treatment had failed suggests that antibacterial treatment should be given to similar cases at the earliest opportunity. The recognition of descending opacities indicates activity, and it is in these patients that the greatest improvement may be expected. A group of older patients with similar radiographic appearances but without serial changes may represent later stages of the same process, but since activity may have ceased, their response to antibacterial treatment is unpredictable. Since the original process appears to begin during adolesence, patients without serial changes are more likely to benefit from antibacterial therapy if they are young than if old.

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Summary

1. Ten examples are described of an uncommon type of symptomless chronic pulmonary tuberculosis, which can be recognised radiographically by fine opacities slowly decending from apices to lower zones over many years.

2. The clinical and bacteriological features of these patients are described, and their response to antibacterial treatment after other methods of treatment had failed is reported.

3. Reasons are given for believing that patients with similar but not identical radiographic appearances may also benefit from antibacterial treatment.

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THE USE OF DIONOSIL FOR BRONCHOGRAPHY IN PULMONARY TUBERCULOSIS

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Introduction

The object of this paper is to give an account of our experiences in the use of Dionosil for bronchography. Dionosil is a suspension of the *n*-propyl ester of 3 : 5-di-iodo-4-pyridone-N-acetic acid in water with carboxy-methyl cellulose added. In the lungs or bronchi the ester undergoes hydrolysis and any that is not coughed up is absorbed and eliminated in a few days. The radio-opaque component of Dionosil is closely related to that of Diodone, which is the di-ethanolamine salt of the same acid.

Sixty cases have been examined by this means; in ten the bronchograms were part of routine diagnostic investigations; the other fifty, with which this report is chiefly concerned, were tuberculous, and the object was assessment for subsequent treatment. Some details are given in the Table.

It is not our purpose here to discuss at length the larger question of the value of bronchography in such cases. The importance of bronchial lesions in pulmonary tuberculosis has been increasingly recognised during the last decade or so; bronchoscopy has become a routine investigation. Bronchography provides a more complete picture of the bronchial tree and may be of particular value at times. The natural reciprocal variation in the size of the broncho-pulmonary segments may make the exact location of a lesion uncertain and distortion due to past or present collapse therapy may contribute to the difficulty. Bronchial anatomy may vary from the usual pattern, especially in the left upper lobe (Foster-Carter, 1952). Where a diseased or retracted upper lobe is present the position and integrity of the apical segment of the lower lobe may be difficult to determine by any other means.

Cases 1 to 5 will serve as examples of these problems and as samples of the quality of the bronchograms obtained with Dionosil.

Case 1. J.W., aged 31, had bilateral disease in 1942; he was well and working between 1943 and 1952, when an X-ray showed increasing disease and sputum culture was positive. The bronchogram (Fig. 1) shows that the bronchi to the anterior segment of the left upper lobe and to the lingula have a common origin.

Case 2. M.A., female aged 23, developed tuberculosis at the age of 18 years. On admission, in January 1952, the sputum was positive and X-rays suggested bronchiectasis rather than pulmonary cavitation. Bronchograms (Fig. 2 and 3) showed marked bronchiectasis of the right upper lobe, but a normal bronchial tree in the apex of the lower lobe and remaining segments of the lung.

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CASE 3. R.B., aged 20, had a left artificial pneumothorax in 1951, and in 1952 returned to hospital with a fresh lesion on the right side. A right artificial pneumothorax was induced but the sputum remained positive. Bronchograms (Fig. 4) showed a block in a branch of the apical segmental bronchus related to the lesion.

CASE 4. R.C., aged 24, was found to have pulmonary tuberculosis in 1950, and in 1952 a fresh lesion developed on the left side. The extent of the disease was difficult to ascertain, but bronchograms (Fig. 5) showed bronchiectasis involving the apical segment of the lower lobe as well as part of the upper lobe.

CASE 5. P.S., female aged 20, had a persistent cough and wheeze for six months before pulmonary tuberculosis was diagnosed in October 1951. On admission in May 1952, X-rays showed atelectasis of the left lower lobe. Bronchoscopy revealed stenosis of the left main bronchus sufficient to prevent any examination below its first half-inch or so. Bronchograms (Fig. 6) show the lower lobe bronchus completely occluded except for its apical branch, a finding confirmed later at operation.

Technique

Any series of bronchograms must depend for its success upon satisfactory posturing and upon good radiography, both of which must be adapted to the medium in use. It may be that all the techniques commonly used for bronchography will be suitable for Dionosil. We have used a Jacques catheter (No. 5 English) passed through one nostril into the trachea. Certain points we feel to be of importance and would like to stress:

(*a*) Dionosil provokes coughing more readily than oil, and good anæsthetisation is essential. We give 3 grains of sodium amytal about half an hour before starting, and immediately before proceeding the patient sucks one lozenge of Amethocaine 0·1 g. Anæsthesia is completed by injecting either 2 ml. of 4 per cent. xylocaine or 0·6 ml. of 10 per cent. cocaine with a fine needle through the cricothyroid membrane. This is the same anæsthetic that we have used for eighteen months for bronchoscopy and no reaction has been attributed to it.

(*b*) The suspension requires very thorough shaking before use. It should also be warmed to approximately blood heat; this is a great help in reducing coughing. It does not, of course, as in the case of Neohydriol, lead to significant reduction in viscosity.

(*c*) Dionosil flows more freely than oil, and it must not be injected too quickly as this will lead to coughing. Using a tilting table with an under-the-table tube, the patient lies on the side to be examined and 3-4 ml. of Dionosil is injected in about one minute; appropriate posturing then directs it into the required area; 10-12 ml. usually suffices to fill one lung. Filling is watched fluoroscopically, and films are then taken in the erect position. The patient lies again on the appropriate side while they are developed, as Dionosil tends to run out of the upper bronchi.

(*d*) Dionosil contains 30 per cent. of iodine and Neohydriol 40 per cent.; exposures must be modified accordingly. Spencer-Jones (1952), writing of Umbradil-Viscous-B, has indicated that it is not sufficiently radio-opaque to give good bronchograms. We have found, however, that the related Dionosil gives films perfectly satisfactory in this respect.

PLATE V

FIG. 1.—The bronchi to the anterior segment of the left upper lobe and the lingula arise by a common origin from the left upper bronchus.

FIGS. 2 and 3.—There is bronchiectasis of all upper lobe segments. The apical segment of the lower lobe is clear.

FIG. 4.—There is a block in one division of the apical bronchus which is related to the parenchymatous lesion.

FIG. 5.—There is bronchiectasis of the upper lobe and the apical segment of the lower lobe.

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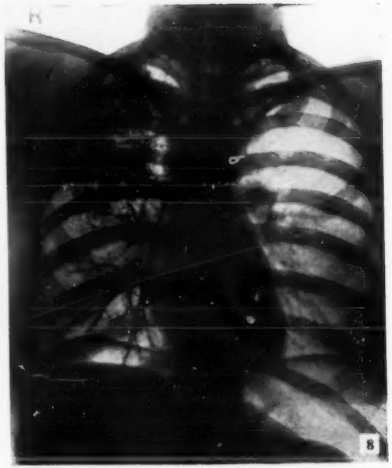
PLATE VI



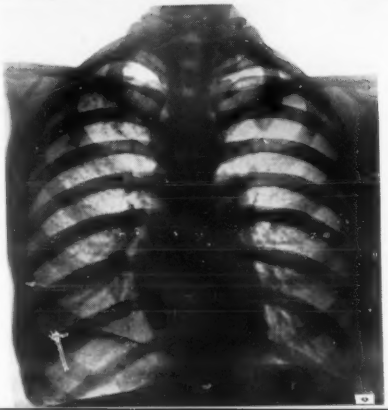


FIG. 6.—Complete stenosis of the left lower bronchus. There is also partial stenosis of the left main bronchus.

FIG. 7.—There is stenosis of the left upper bronchus and bronchiectasis in the lower lobe.

FIG. 8.—Shows a block in one branch of the posterior upper lobe bronchus.

FIG. 9.—A film taken five days after bronchography showing a little residual Dionosil. Three days later none remained.

Fig. 7 is a bronchogram of a woman who had undergone a left thoracoplasty; although most of the left lung field lies behind the heart and the pleura is thickened, the bronchial tree shows plainly.

Discussion

Iodized oils have been used for some thirty years in bronchography. They have certain disadvantages:

(*a*) The oil is liable to remain in the alveoli or finer air passages or elsewhere, and there to throw shadows for an indefinitely long time. These may obscure clinically significant shadows, and may on occasion lead to a mistaken diagnosis. Iodised oil is included in a list of the causes of chronic diffuse nodular or reticular shadows in the lungs given by Scadding (1952).

(*b*) Alveolar filling may occur before films have been taken and may obscure the bronchial anatomy.

(*c*) There is some evidence that after lobectomy atelectasis of the residual lobe is more likely to occur if bronchography with iodised oil has been done shortly before operation (within two months). Some surgeons at least have remained unwilling to operate within such an interval since Belsey (1937) recorded his findings in the comparatively early days of dissection lobectomy.

(*d*) Acute iodism is a rare but serious complication of the use of iodised oils. It may be avoided by preliminary tests, but the bronchogram is left undone.

(*e*) It is doubtful whether residual oil produces an inflammatory reaction or activates a quiescent tuberculous lesion or not. Pagel *et al.* (1948) indicate this doubt. Young and Ganz (1941), examining lobes removed surgically which contained residual oil, found evidence of chronic alveolar and bronchiolar inflammation which appeared to be associated with the oil. They also showed that Neohydriol kept in the presence of sputum, or in a damp atomosphere in the dark and at room temperature, underwent a chemical change greater than if it was kept dry but in otherwise similar conditions.

Marchese *et al.* (1952) studied forty-nine patients with pulmonary tuberculosis in whom Lipiodol bronchograms were performed; spreads of disease occurred in only three patients, and in these only after six weeks or more; all of them had shown similar spreads during the four months before investigation. Reviewing the literature, these authors mention a number of papers, some of them dealing with large series of cases, in which there are few reports of any bronchogenic spreads.

Felton (1953) made a histological study of thirty-seven resected lung specimens from patients who had been examined by Lipiodol bronchography between three weeks and forty-seven months before operation. Twenty-three of them contained residual Lipiodol, and all of them showed bronchiectasis and areas of organising pneumonitis. In eight Lipiodol was concentrated in these areas, and in a number focal foreign-body granulomata were seen.

Whatever may be the truth about the effect of Neohydriol or similar preparations upon healthy or tuberculous bronchi or lung tissues, many, in this country at least, have been reluctant to use them in cases of tuberculosis. The most important drawback is the persistence of the oil and of the shadows which it casts.

F. H. Young (1953) has remarked upon the need for some contrast medium other than Neohydriol, and suggested that the sulphanilamide-lipiodol mixture reported upon by Houghton *et al.* (1951) might be suitable. We used this mixture in two cases and obtained good bronchograms; but both patients developed methæmoglobinæmia and we therefore awaited further reports upon it; meanwhile, Dionosil became available.

Spencer-Jones (*loc. cit.*) reported unfavourably upon the use of Umbradil-viscous-B; he found that it proved so irritating that he had to abandon the attempt in five out of nine cases, and in only two did he obtain satisfactory films. Morales (1949) considered it a suitable medium for the purpose and superior in its physical properties to Neohydriol. He states that the procedure is " fairly tiresome " for the patient. Morales and Heiwinkel (1948) considered it non-irritating and obtained good results in a few cases. Atwell and Pederson (1950) used it for an unspecified number of cases and thought that its advantages outweighed its disadvantages.

Don (1952) used Dionosil for a series of sixty bronchograms in adults; after a satisfactory technique had been worked out he obtained uniformly good results. His object was much the same as ours—*viz.*, pre-operative assessment. We cannot, however, agree with his finding that a catheter technique is unsuitable for Dionosil; and we do not use such heavy premedication as he found necessary. Our apparatus does not permit us to take satisfactory films in recumbency, and it has therefore been essential for our patients to be capable of standing up and walking.

Rendle-Short (1953) found Dionosil satisfactory for bronchography in children under general anæsthesia.

Our results with Dionosil, paying particular attention to the drawbacks of iodised oil mentioned above, are now considered.

In column 3 of the table we have indicated the degree of filling achieved. Our aim has been to fill all the bronchi in the part under examination at least as far as all the recognised sub-segmental branches. Where we have described the filling as good this has been approximately attained and the films have been technically satisfactory.

Thirteen cases were regarded as adequate (two non-tuberculous cases are not in the table). These achieved their object by demonstrating the state of the bronchi under consideration, but did not reach the above standard. In most, filling was somewhat incomplete, and in a few, indifferent radiography and accidental filling of the opposite side contributed. Coughing and intolerance of the procedure were the principal difficulties encountered and were also to blame for three of the four failures (two not in the table). The fourth was largely due to poor positioning, but here too coughing was very troublesome.

Others with whom we have talked have also found that Dionosil often provokes coughing. We do not, however, consider that coughing is so frequent or so severe as to condemn the medium. There will always be a small proportion of patients who are intolerant of any interference with their respiratory tract.

Dionosil in the quantities in which we have used it tends to line rather than fill the bronchi. When coughing occurs, some is often coughed up and swallowed, but the opaque lining remains. Don (*loc. cit.*) found the same. Excessive filling, if it occurs, gives a much less dense and woolly appearance

than happens with Neohydriol, and has never obscured the bronchial tree. Some excessive filling was recorded in seventeen cases, but in ten of these it was trivial. Case 7 referred to below is an example of what we imply by excessive filling.

Dionosil disappears rapidly. Column 5 in the table gives some details. After the first few cases we were satisfied that Dionosil does not, in fact, persist to any important extent or for any length of time, and most subsequent cases were not, therefore, X-rayed again until this became due as a routine. Of those X-rayed after a short interval traces of Dionosil were visible after one day in twelve cases and after a rather longer time in seven others. The residuum was trivial and in no way comparable to the heavy shadows often left by Neohydriol. For practical purposes Dionosil disappears in a few days.

Case 7. J.W., female aged 25, had been intermittently under treatment since 1949. A bronchogram was done on March 9, 1953. She coughed once as the first bolus of Dionosil reached the trachea, thereafter she did not cough. The film (Fig. 8) shows the type of excessive filling that we have encountered. It does not obscure the block in one branch of the posterior segment. The lateral film taken at the same time was technically unsatisfactory and she coughed repeatedly before another could be taken, but no further excessive filling occurred. The Dionosil persisted longer than usual. Fig. 9 shows the appearance five days later; none was visible three days after this.

We have considered as an inflammatory reaction any pyrexia or increase in pyrexia occurring after the bronchogram, and any increase in radiological shadowing not directly attributable to the contrast medium. All patients listed in the table have been under observation at least one month after bronchography.

In a report by Houghton and Joules (1953) on an investigation into tuberculous tracheobronchitis, a rise of temperature above 99° F. in the three days after bronchoscopy in a patient normally afebrile (*sic*) was taken as a sign of a complication; they found such a rise in only 3 of 251 patients. In 103 tuberculous patients bronchoscoped here in the last twelve months the incidence of slight fever has been rather higher. Eight patients have shown such a rise of temperature. Of the 50 patients in the table, 17 were febrile after bronchography; 40 were also bronchoscoped, and among them fever occurred in 15 after bronchography, and in only 2 after bronchoscopy.

In 12 of these 17, however, the reaction was very slight—*viz.*, a rise in temperature on the first or second evening which returned to its normal level in a few days, four in one case and less than four in the remainder. The temperature rose to between 99·2° and 100° F. in eight cases and between 100·2° and 101° F. in four. Most of these patients had a slight increase in cough and sputum for a few days, as did a number of others, but in no case did sputum become positive where it had formerly been negative for three months. Don (*loc. cit.*) found a comparable number of pyrexial reactions—twelve in forty-five cases.

Six cases require further consideration:

(1) W.R. male aged 24, had a temperature of 101·2° F. the next evening, and of 98·4° F. after forty-eight hours, when he was well. Sixteen days later he

developed a " cold " with slight fever and increase in cough and sputum and a film ten days after this showed a slight increase in the size of his cavity. There was an epidemic of such colds at the time, and we believe that it was due to this and not to the bronchogram.

(2) H.T., male aged 26, had a temperature of 100·2° F. for one night. He then remained apparently well but three weeks later a film showed some increase in a diffuse lesion in the left upper lobe. He was given Streptomycin, Isoniazid and Paraminosalicylic acid and in a further month his condition had returned to its former state. Throughout there was no further pyrexia and no cough or sputum.

(3 & 4) Two men, R.S., aged 32 and A.B., aged 21, had attacks of bronchitis, with typical symptoms, developing two days after bronchography. Neither showed any radiological change and in both the attack abated quickly when treated with penicillin. R.S. has lower lobe bronchiectasis as well as tuberculosis and is subject to such attacks. His temperature was raised only to 99·2° F. In A.B. it reached 103·2° F., but he recovered completely in ten days.

(5) G.W., female aged 37, had severe chronic disease in the left lung with a large upper lobe cavity and diffuse disease elsewhere; the object of the investigation was to determine whether there was bronchiectasis in the lower lobe. The temperature rose to 102·2° F. that night and the sputum, which was positive and remained so, increased considerably for two days. The temperature returned to normal after two days and there was no change in the X-ray appearance.

(6) The final case occurred in a girl, J.H., aged 20, who had a chronic lesion involving the apex of the right lower lobe and the posterior upper lobe segment above it; there was bronchiectasis in both these segments. There was no immediate reaction to the bronchogram, but a film taken eight days later showed a fresh lesion—a homogeneous wedge-shaped opacity involving the apex of the lower lobe. There was no rise in temperature nor increase in sputum and no special treatment was given. Six days later the opacity was less, but two weeks after this she had, for one night only, a temperature of 100·4° F., and as soon after this the opacity was found to have increased again penicillin was given for six days. A week later it was diminishing, and in a fortnight the X-ray appearance had reverted to its previous state.

A series of fifty cases is hardly sufficient to permit a fair judgment upon the liability of a new preparation to provoke inflammatory reactions. Of the six complications here described, one—the first—was probably not a sequel of the bronchogram. Two others—3 and 4—consisted of an acute bronchitis which soon recovered, one being in a subject with non-tuberculous bronchiectasis. It is possible that these may have been a result of nasal secretion carried by the catheter into a trachea which remained anæsthetic for a few hours, but such nasal secretions will probably pass into the trachea and bronchial tree during sleep without any particular provocation. The fifth case was comparable to the other milder responses which we have mentioned and occurred in a patient with a grossly distorted and bronchoscopically inflamed bronchial tree. Cases 2 and 6 were the only ones which showed any radiological change; the first of these was slight and soon recovered with streptomycin and chemotherapy. The last case appears to have been of the nature of a non-specific aspiration pneumonia.

To demonstrate convincingly the presence or absence of any specific

(*Continued on p.* 43).

Age and Sex	*BRONCHOGRAM*				*Reactions*	*Remarks*
	Side	*Filling*	*Clearing*	*Findings*		
R.P. F. 29	L	Good	Complete within 3 weeks	Basal bronchiectasis; obstruction of U.L. bronchus	None	Bronchial condition confirmed after resection (see Fig. 7).
R.K. M. 17	L	Good	Almost complete in 24 hours	Bronchiectasis of postero-apical segment of U.L.	None	
L.B. F. 19	R	Good	Some still present after 24-hours. Complete within 2 weeks	Minimal bronchiectasis in U.L.	Pyrexia to 99·6° F. for 24 hours	
R.B. M. 28	L	Good	Almost complete in 24 hours	Bronchiectasis of lingular bronchi only	None	
R. B. M. 28	R	Adequate	Almost complete in 24 hours	Bronchiectasis of upper lobe and apical segment of lower lobe	None	Old, partially obliterated pneumothorax with chronic empyema. One middle lobe segment unfilled.
M. H. M. 32	R U.L. only	Adequate	Almost complete in 24 hours	Normal	None	Anterior segment not well filled.
M. A. F. 26	L	Inadequate	Trace still present after 3 days; complete within 7 days	Not sufficiently demonstrated	Pyrexia to 100·4° F. — normal after 2 days	A patient with slight disease, but very intolerant; posturing was chiefly at fault.
G. D. M. 21	R	Good	Almost complete in 24 hours; complete within 4 days	Minimal bronchiectasis of apical segment of U.L.	None	
P. S. F. 21	L	Adequate	Complete within 3 weeks	U.L. normal partial stenosis of main bronchus—occlusion of L.L. bronchus	None	Findings confirmed at operation. Filling classed as adequate, as postero-apical segment was incompletely filled (see Case 5).
B. H. F. 31	R U.L. only	Adequate	Complete within 3 weeks	Bronchiectasis all segments	None	Posterior segment poorly filled, but film adequate for its purpose.
R. B. M. 21	R	Good	Complete within 2 weeks	Block in posterior segment of U.L.	None	R.A.P. present (see Case 3).
W. Y. M. 34	R	Good	Complete within 4 weeks	Normal	None	
M. A. F. 24	R	Good	Minute trace only remained after 3 weeks	Bronchiectasis all segments of U.L.	Pyrexia to 99·2° F. for one night	Apical lower lobe segment normal (see Case 2).

Age and Sex	*BRONCHOGRAM*				*Reactions*	*Remarks*
	Side	*Filling*	*Clearing*	*Findings*		
A.C. M. 38	R	Good	Complete within 2 weeks	Normal	None	
A. B. F. 25	R & L	Good	Complete within 2 days	Bronchiectasis of apical segment of R.U.L., and postero-apical segment of L.U.L.	None	
J. W. M. 31	R & L	Good	Complete within 3 weeks	Minimal bronchiectasis of apical segment of L.U.L.	None	See Case 1.
J. W. F. 25	R	Good	Trace remained on 7th day and complete clearing within 8 days	Bronchiectasis and obstruction of one sub-segment of posterior U.L. bronchus	Pyrexia to 100° F. for 24 hours	Bronchoscopy next day showed normal bronchial mucosa (see Case 8).
B. K. M. 23	L	Good	Trace remained after 2 days	Normal bronchial tree. Dionosil entered a small "solid focus"	Pyrexia to 101° F. lasting 3 days	See Case 9.
S. K. F. 19	L	Good	Complete within 24 hours	Bronchiectasis of postero-apical segment of U.L.	None	
H. H. M. 24	L	Good	Complete within 3 weeks	Partial stenosis and bronchiectasis of L.L. bronchi	None	Previous 7-rib thoracoplasty.
R. H. M. 25	L	Adequate	Complete within 7 days	Distortion and bronchiectasis of U.L.	None	Filling of U.L. is incomplete but shows lesion — radiography is poor and apical segment of L.L. not adequately displayed.
B. H. F. 32	R	Good	Complete within 3 weeks	Bronchiectasis of all segments of U.L.	Pyrexia to 99·2° F. for 2 days	No pyrexial reaction after previous bronchogram (of U.L. only).
J. H. F. 20	R	Good	Complete within 8 days	Bronchiectasis of posterior segment of U.L. and apical segment of L.L.	No pyrexia. Fresh pneumonic lesion visible on X-ray after 8 days	See text.
R. C. M. 24	L	Good	Complete within 7 days	Bronchiectasis of posterior segment of U.L. and apical segment of L.L.	None	See Case 4.

Age and Sex	*BRONCHOGRAM*				*Reactions*	*Remarks*
	Side	*Filling*	*Clearing*	*Findings*		
F. G. M. 38	R	Adequate	Trace remained after 2 days	Normal	None	Posterior segment of U.L. and apical segment of L.L. incompletely filled.
D. G. M. 28	L	Good	Almost complete in 24 hours	Normal	None	
L. B. M. 21	L	Good	Trace remained after 24 hours. None after 4 days	Bronchiectasis of left upper lobe except lingula	Acute bronchitis with pyrexia to 103° F.	See text.
T. W. M. 34	L	Good	Complete in 24 hours	Normal	None	
R. P. M. 33	L	Adequate	Complete within 4 weeks	Slight bronchiectasis and marked distortion of upper lobe bronchi	Pyrexia to 99·4 F. lasting 2 days	Basal bronchi incompletely filled. Film difficult to interpret owing to distortion, poor radiography and filling of opposite side.
S. A. F. 21	L	Good	Complete within 1 week	Normal	Pyrexia to 100° F. lasting 24 hours	
S. M. F. 19	L	Good	Complete within 1 week	Normal	None	
G. W. F. 30	L	Adequate	Trace remained after 2 days	Gross distortion and bronchiectasis of upper lobe, with open pulmonary cavities	Pyrexia to 102·2° F. lasting 2 days	Dionosil flowed out of upper lobe bronchi and provoked coughing whenever the patient sat up.
HMcK M. 28	L	Good	Complete within 3 weeks	Bronchiectasis of upper lobe bronchi	None	
E. B. M. 21	R	Good	Trace remained after 2 days	Normal	None	R.A.P. present.
R. F. M. 19	L	Good	Almost complete in 24 hours	Bronchiectasis of apical segment of L.L.	None	Previous L. upper lobectomy and thoracoplasty.
B. P. M. 42	L	Good	Almost complete in 24 hours	Obstruction in one branch of apical segment of U.L.	None	
A. L. M. 33	R	Good	Complete within 3 days	Slight bronchiectasis of a contracted L.L.	None	
E. P. M. 26	L	Inadequate	Complete within 4 weeks	Bronchiectasis of U.L. bronchi	Pyrexia to 100·4° F. lasting 4 days, with radiological increase of lesion	See text. Incomplete filling of both lobes.

Age and Sex.	*BRONCHOGRAM*				*Reactions*	*Remarks*
	Side	*Filling*	*Clearing*	*Findings*		
G. M. F. 39	L	Good	Complete within 8 days	Bronchiectasis upper and lower lobes	None	Previous 8 rib thoracoplasty.
M. C. F. 37	R	Good	Complete within 8 days	Normal	Pyrexia to 99·6° F. for 2 days	
A. N. M. 24	R	Good	Complete within 10 days	Bronchiectasis apical and posterior segments of upper lobe	None	
W. R. M. 24	L	Adequate	Complete within 4 weeks	Distorted; not bronchiectatic	Pyrexia to 101·2° F. lasting 24 hours only	One segment of lingula not filled. Previous pleural effusion.
W. H. M. 17	R & L	Adequate	Complete within 4 weeks	Bronchiectasis of both upper lobes	Pyrexia to 100·2° F. lasting 24 hours	Middle lobe incompletely filled.
S. C. M. 21	L	Good	Complete within 1 week	Bronchiectasis (slight) of postero-apical segment of U.L. bronchus.	None	Previous 8 rib thoracoplasty.
D. G. F. 24	L	Good	Complete within 1 week	Normal	None	Already pyrexial.
R. M. M. 29	L	Good	Complete within 1 week	Normal	None	
R. S. M. 32	R & L	Good	Complete within 3 weeks	Bronchiectasis of posterior segment of upper lobe and of apical, anterior basal, and medial basal segments of lower lobe	Acute bronchitis pyrexia to 99·2° F.	See text.
H. G. M. 31	L	Good	Complete within 8 weeks	Bronchiectasis of apical segment of lower lobe and of lingula	None	Previous 7 rib thoracoplasty.
D. O. M. 22	L	Good	Unknown (temporarily discharged)	No bronchiectasis	Pyrexia to 99·8° F. lasting 24 hours	Previous 9 rib thoracoplasty.
E. E. F. 33	L	Adequate	Complete within 16 days	Stenosis of left main bronchus; occlusion of upper lobe bronchus	Pyrexia to 99·2° F. lasting 24 hours	

response to a contrast medium has proved difficult in the case of iodised oils and may be more so with the much less persistent Dionosil; subjects vary greatly in the readiness with which their bronchi develop slight reactions to any mild stimulus. The earliest and least reaction is an increase in the mucous secretion, and hyperæmia, leading, if sufficient, to some cough and mucoid sputum; such changes quite commonly occur after bronchoscopy or the passage of an intra-tracheal tube and it is reasonable to expect them after bronchography with any medium.

Hellström and Holmgren (1949) studied the effect of intra-bronchial injection of physiological saline into the bronchi of white rats and rabbits, and found that the lungs showed thickening of the alveolar septa, increase in blood content and increase in size of the alveolar phagocytes; and that these changes were of the same nature as, though less in degree than, those which occur with Umbradil, Umbradil-Viscous-B, and carboxy-methyl-cellulose; they considered the changes to be non-specific.

In a bronchus which is already inflamed such changes will tend to be more marked and the bronchus will be more readily occluded; secretion will be likely to contain tubercle bacilli and tuberculous products and, if retained, will tend to set up the changes which occur with infected atelectasis; or, if aspirated into other parts of the lung, may cause fresh tuberculous foci; but if the inflammation is not severe or continuous, these changes are not likely to be marked or irreversible.

If bronchography is to be carried out at all in such cases it seems probable that such complications must be expected occasionally, but they are of a type which will generally respond readily to streptomycin, other antibiotics or chemotherapeutic drugs. Though we have not made a practice of it in this series, it appears to us that it might be wise to give streptomycin for a short time where a temperature occurs after an investigation. Obviously one would avoid carrying out bronchograms on patients with active or severe disease so far as possible, but this is not always feasible. Few of our patients had had less than six months' bed rest and some much more, and many of them had little prospect of becoming quiescent without operation.

Finally, no reactions comparable to acute iodism have occurred; no free Iodine is liberated from Dionosil; the related Diodone has been in use for many years and toxic or hypersensitive reactions to it are said to be extremely rare.

Summary and Conclusions

1. Dionosil, a watery suspension of an organic iodine compound containing 30 per cent. of iodine, has been used in a series of bronchograms, including fifty carried out on patients with pulmonary tuberculosis. Some points in the technique used are mentioned.

2. The value of bronchograms in assessment of such cases for treatment is briefly considered.

3. Complications arising after bronchography in these fifty patients are discussed. Seventeen patients (34 per cent.) showed a febrile reaction, in most cases slight. Six cases, including five of the above seventeen, had some more notable complication, but in none was it serious or lasting.

4. Dionosil does not persist in the lungs or bronchi, and excessive filling has not occurred to an extent which renders interpretation of the bronchograms difficult. In these respects it compares favourably with iodised oils. Its principal drawback is its tendency to cause coughing. With adequate anæsthesia, however, this leads only occasionally to failure. Two of our four failures occurred in the first ten cases investigated.

5. Dionosil is considered to be a more valuable agent for bronchography, in tuberculous patients at least, than iodised oils.

We are very grateful to Dr. A. K. Miller for his interest in and help with this paper; and to Mr. E. F. Chin who suggested to us the value of bronchography in these cases of tuberculosis, and who first introduced us to Dionosil. We also wish especially to thank Mr. V. J. B. Higgins, our radiographer, for his painstaking and enthusiastic help with the bronchograms.

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ARTIFICIAL PNEUMOPERITONEUM

WITH PARTICULAR REFERENCE TO ITS VALUE IN CAVITY CLOSURE

A REVIEW OF A PERSONAL SERIES

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Pneumoperitoneum has been used therapeutically since 1872, for conditions as diverse as tuberculous peritonitis and gastric hæmorrhage. However, it was not until 1933 that Vadja reported its use for the treatment of pulmonary tuberculosis. Later in 1933 Banyai reported that in 1931 he had injected air accidentally under the diaphragm when inducing an artificial pneumothorax to control a massive hæmoptysis due to pulmonary tuberculosis. In 1935 Klopstock and Scheuler combined pneumoperitoneum with phrenic nerve paralysis for the first time. Its use has been disappointing in advanced pulmonary cases, the improvement in these being mainly symptomatic and of little permanent value (Daniels and Eisele, 1938). During recent years a more scrupulous selection of cases has improved results, but even so this treatment has been criticised (Hurst, 1947) and even dismissed by some physicians (Naegeli, 1948; Chatard, 1948).

Indications

Earlier reports stressed the general use of this treatment, whereas later papers concentrated on defining the exact intrapulmonary lesions, where pneumoperitoneum appeared to be indicated. The closure of cavities in relation to their nature and position in the lungs was particularly studied.

Rilance and Waring (1941 and 1944) had greatest success in treating lower and mid-zone cavities, while Clifford-Jones and Macdonald (1943) found upper and lower zone cavities more responsive than those in the middle zone. Peter Edwards and Logan (1945) concluded that " in the presence of adequate rise of the diaphragm it is of minor importance where the cavity is situated, but of major importance whether the disease is of the soft recent type or the long-standing fibrous type; and whether cavities, if present, are extensively adherent or surrounded by relatively normal lung." Keers (1948) agreed with this view, and advised its use for contracting fibrotic " lesions " also.

Banyai (1946) found that cavities in the lower lobe or middle third of the lung were more responsive. Similar views were expressed by Lyons (1949) and also Rosenblat (1949). Frazer (1950) found that pneumoperitoneum helped patients to thoracoplasty and reduced the extent of the operation. Thompson Evans (1950) found middle and lower zone cavities more responsive to those in the upper zone. Habeeb and Reisner (1950) stressed the

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importance of the character of the cavity wall and surrounding disease in determining successful closure. Basal lesions were particularly responsive. Netzer (1951), reviewing the long-term results of pneumoperitoneum, found greater success with exudative lesions as opposed to fibrotic lesions. This view was repeated by Crenshaw and Gross (1952).

Contra-indications

Contra-indications, for practical purposes, are few, and although Banyai lists eight general indications, most of them are in themselves clearly evident. He also states that pneumoperitoneum is contra-indicated when the hemidiaphragm is fixed by pleural adhesions. This fact can be ascertained beforehand on a double-exposure radiograph taken on full inspiration and expiration. The limits of movement of the diaphragm can thus be permanently recorded. The presence of plastic peritonitis is a contra-indication, because bowel adhesion to the abdominal wall increases the risk of bowel perforation by the refill needle. Hermandez *et al.* include acute inflammatory conditions of the abdomen, as pneumoperitoneum would hinder localisation of the inflammation by peritoneal adhesions. It is of interest that Rilance and Waring found appendicitis more common in their series of pneumoperitoneum patients. They stated that pelvic inflammatory conditions should contra-indicate this treatment for the same reason. A previous history of inflammatory abdominal conditions indicates the possibility of peritoneal adhesions which may be responsible for abdominal pain during treatment.

Complications and Dangers

Clifford-Jones and Macdonald noted that vomiting was uncommon in their series, but occurred when the tendency to this was already present. Bowel perforation occurred once in their series, as also did peritoneal effusion. Banyai considers that the longer a pneumoperitoneum is maintained the more likelihood there is of peritoneal effusion and of the development of tuberculous foci on the peritoneum. Peter Edwards and Logan reported four small peritoneal effusions in fifty pneumoperitoneums, one of which developed a fulminating tuberculous peritonitis. The remainder absorbed slowly and did not interfere with treatment. E. L. Williams noted this complication in 2·6 per cent. of his cases and Frazer in 5 per cent.

Air embolism has been reported by Fremmel as occurring once in 200 cases. Bailey records a fatal case occurring at the first refill. Post-mortem examination showed air bubbles in the right side of the heart. Sita-Lamsden with Elphinstone, and also Hollander, have reported this complication occurring in their experience. Batson stated that free anastomosis occurred between the abdominal veins and the vertebral venous plexus, and suggested this was a possible route for cerebral embolism occurring during pneumoperitoneum treatment.

Accidental pneumothorax can occur when refills are given through the lower intercostal spaces and by the subdiaphragmatic route. Rupture of the diaphragm can be responsible for this condition and has been reported by Wynn-Williams.

Mediastinal emphysema was experienced by Banyai in five of his cases. Air escaped through a congenital or acquired defect in the peritoneum or through the diaphragmatic hiati and reached the mediastinum. Tinker has also reported this complication in his experience. Klopstock and Schueler produced emphysema of the omentum in one patient during a refill. Subcutaneous emphysema is very common and must have been encountered by everyone.

Herniæ of the anterior abdominal wall have been reported by Banyai, but all were controlled satisfactorily by an abdominal binder. He encountered three umbilical herniæ and one scrotal pneumocele in his series.

Stokes observed " atelectasis " in two of his cases and said it was due to interference with bronchial drainage. Banyai also noted this in a patient with extensive endobronchial tuberculosis. He encountered two cases of diarrhœa, but no evidence of tuberculous bowel infection could be found, and they responded to medical treatment.

In this series very few complications were encountered.

TABLE I

Sectional collapse of lower lobe	1 case
Peritoneal effusion	2 cases
Umbilical pneumocele	1 case

Sectional collapse of the right lower lobe was encountered in one case (No. 11), where the right hemidiaphragm was elevated by 4 inches. The frequency and volume of the refills were reduced and the collapsed area soon expanded.

Peritoneal effusion occurred in two cases (Nos. 8 and 14). No intervention was made in these because there was no distress. Refills were continued but less air was required to maintain the usual degree of elevation of the diaphragm. The effusion in one case (No. 9) absorbed in three weeks, meanwhile refills were maintained. In the other case (No. 14) the effusion was small and did not affect treatment.

Pneumocele of the umbilicus occurred in one case (No. 24). The patient was a married woman aged 42 years, who had had five pregnancies. There was slight devarication of the recti muscles. The swelling responded to local pressure and decreasing the frequency and amount of refills. These were the only complications encountered in this series.

TECHNIQUE AND MANAGEMENT.

The technique of pneumoperitoneum treatment has been modified by individual physicians as a result of their experience, although the basic technique has remained the same.

One aspect, on which there is not complete agreement, is the site of insertion of the refill needle. The writer prefers to insert the needle about 1½—2 inches above and to the left of the umbilicus. For safety's sake, the tip of the left thumb is placed in the umbilicus and the abdominal wall on the opposite side is gripped between it and the left index finger. The abdominal wall is then pulled upwards, thus raising it away from the intra-abdominal viscera. The refill needle is introduced in the above position at an angle of

40 degrees to the horizontal. This greatly reduces the risk of injury to abdominal viscera and so gives increased confidence with this treatment. After air has been introduced, the needle is removed, the abdominal wall is released, and the needle track is readily obliterated.

In this series, where the object of pneumoperitoneum treatment was cavity closure, the writer found that the following scheme of treatment was to be recommended. At induction 1,000 c.c. of air were given, and in the absence of any untoward symptoms were followed by 2,000 c.c. twice weekly until adequate elevation of the homolateral hemidiaphragm was visualised on radioscopy. The refills were then reduced to 2,000 c.c. once weekly. These large refills early in treatment produced rapid elevation of the diaphragm resulting in maximum effect on intrapulmonary mechanics. Cavity closure, whether by approximation of walls or obliteration of draining bronchi, was in that way more probable and occurred as early as possible, thus eliminating quickly the inherent dangers of a patent cavity. Good elevation of the diaphragm was maintained for the whole period of treatment, and care is particularly needed when the frequency of refills is being reduced. It was noted that if the cavity reopened during that period it was unlikely to close again by the use of pneumoperitoneum.

These large refills were reasonably well tolerated. A feeling of distension in the upper abdomen occurred after the early refills, but this disappeared in about two hours and later was not noticed. Dyspnœa after refill was uncommon and was only particularly noticeable in one patient, where respiratory function was already impaired by a contralateral partially functioning hemidiaphragm. She was able to walk home and dyspnœa disappeared in a few hours.

" Ballottement " of cavities, particularly the tension variety, by alternately raising and lowering the diaphragm by decreasing and increasing the amounts of refills, should always be tried before concluding that the pneumoperitoneum is unsuccessful.

Results

In this series it is notable that in ten of the successfully treated cases cavitation was situated in the lower lobe, in fourteen cases it was in the upper lobe,

Table II

Position in lungs	*Successful*	*Unsuccessful*
Upper lobe	14	6
Lower lobe	10	3
Upper and lower lobes	3	4

Table III

Position in lung	*Right lung*		*Left lung*	
	Successful	*Unsuccessful*	*Successful*	*Unsuccessful*
Upper lobe	7	3	8	3
Lower lobe	6	3	4	–
Upper and lower lobes	3	2	–	2

SUCCESSFUL CASES

Case No.	*Age & sex*	*Type and position of disease*	*Initial sputum*	*Final sputum*	*Elevation of diaphragm on phrenic side*	*Treatment*
1	28 F	Small ¾″ cavity in right lower lobe	Pos.	Neg.	Right diaphragm 2½″	Right phrenic crush and pneumoperitoneum.
2	28 F	Medium-sized tension cavity (1½″) base of right lower lobe	Pos.	Neg.	Right diaphragm 3½″	Right phrenic crush and pneumoperitoneum.
3	30 M	Large tension cavity (3″) in left lower lobe	Pos.	Neg.	Left diaphragm 5½″	Left phrenic crush and pneumoperitoneum.
4	19 M	Small cavity (1″) right lower lobe	Pos.	Neg.	Right diaphragm 2½″	Right phrenic crush and pneumoperitoneum.
5	36 F	Medium-sized cavity (1½″) in left lower lobe	Pos.	Neg.	Left diaphragm 4½″	Left phrenic crush and pneumoperitoneum.
6	25 M	Medium-sized cavity (1½″) apex of right lower lobe	Pos.	Neg.	Right diaphragm 2½″	Right phrenic crush and pneumoperitoneum.
7	30 F	Large tension cavity (2½″) in left lower lobe	Pos.	Neg.	Left diaphragm 2½″	Left phrenic crush and pneumoperitoneum.
*8	17 F	Medium-sized cavity (1½″) left lower lobe	Pos.	Neg.	Left diaphragm 2″	Left phrenic crush and pneumoperitoneum.
9	30 F	Medium-sized cavity (1½″) in right upper and apex of right lower lobe and left upper lobe	Pos.	Neg.	Right diaphragm 3″, left diaphragm 2″	Right phrenic crush and pneumoperitoneum.
10	15 M	Small cavity (¾″) right lower lobe	Pos.	Neg.	Right diaphragm 2½″	Right phrenic crush and pneumoperitoneum.
†11	21 F	Small cavity (¾″) in upper part of right lower lobe	Pos.	Neg.	Right diaphragm 4″	Right phrenic crush and pneumoperitoneum.
12	19 F	Small tension cavity (1″) in right upper lobe	Pos.	Neg.	Right diaphragm 4″	Right phrenic crush and pneumoperitoneum.
13	28 F	Medium-sized cavity (1½″) right upper lobe and bronchogenic disease	Pos.	Neg.	Right diaphragm 3½″	Right phrenic crush and pneumoperitoneum.

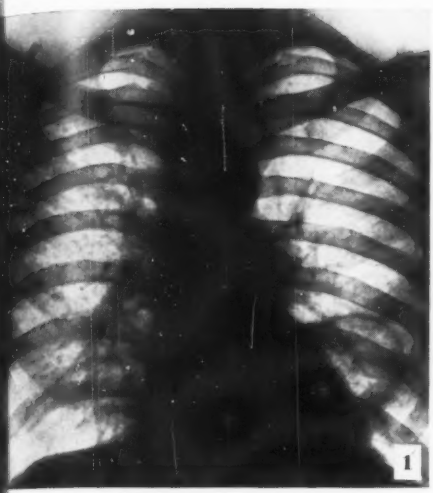
* Complication: Small peritoneal effusion.
† Complication: Sectional collapse of the right lower lobe.

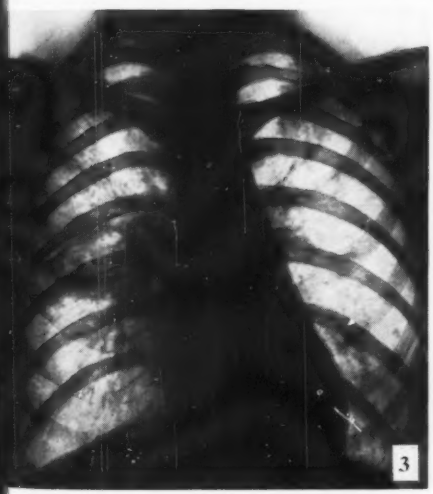
SUCCESSFUL CASES—*Continued*

Case no.	*Age & sex*	*Type and position of disease*	*Initial sputum*	*Final sputum*	*Elevation of diaphragm on phrenic side*	*Treatment*
14	27 F	Small cavity ($\frac{3}{4}$″) right upper lobe	Pos.	Neg.	Right diaphragm 5″	Right phrenic crush and pneumoperitoneum.
15	15 F	Small tension cavity (1″) right upper lobe and bilateral infiltration	Pos.	Neg.	Right diaphragm $3\frac{1}{4}$″, left diaphragm $1\frac{1}{4}$″	Right phrenic crush and pneumoperitoneum
16	21 F	Small cavity (1″) left upper lobe	Pos.	Neg.	Left diaphragm $2\frac{1}{2}$″	Left phrenic crush and pneumoperitoneum.
17	17 F	Medium-sized tension cavity (2″) left upper lobe	Pos.	Neg.	Left diaphragm 2″	Left phrenic crush and pneumoperitoneum.
18	24 M	Medium-sized cavity (2″) left apex	Pos.	Neg.	Left diaphragm 2″	Left phrenic crush and pneumoperitoneum.
19	20 F	Tension cavity (2″) left upper lobe and bronchogenic disease	Pos.	Neg.	Left diaphragm $3\frac{1}{2}$″	Left phrenic crush and pneumoperitoneum.
20	18 M	Medium-sized cavity ($1\frac{3}{4}$″) right upper lobe and cavity ($1\frac{1}{4}$″) right lower lobe	Pos.	Neg.	Right diaphragm 2″	Right phrenic crush and pneumoperitoneum.
21	20 F	Medium-sized tension cavity (2″) left upper lobe and bronchogenic disease	Pos.	Neg.	Left diaphragm $2\frac{1}{4}$″	Left phrenic crush and pneumoperitoneum.
22	22 F	Small cavities in right and left infraclavicular region ($\frac{3}{4}$″) and in apex of right lower lobe (1″)	Pos.	Neg.	Right diaphragm $2\frac{1}{4}$″, left diaphragm 2″ (no phrenic)	Right phrenic crush and pneumoperitoneum.
23	20 M	Small cavity ($\frac{3}{4}$″) right apex	Pos.	Neg.	Right diaphragm $2\frac{1}{2}$″	Right phrenic crush and pneumoperitoneum.
*24	42 F	Small tension cavity (1″) right upper lobe	Pos.	Neg.	Right diaphragm 3″	Right phrenic crush and pneumoperitoneum.
25	16 M	Two cavities left upper lobe, one tension ($\frac{1}{2}$″-1″)	Pos.	Neg.	Left diaphragm $3\frac{3}{4}$″	Left phrenic crush and pneumoperitoneum.
26	28 F	Tension cavity ($1\frac{1}{2}$″) left upper lobe	Pos.	Neg.	Left diaphragm 2″	Left phrenic crush and pneumoperitoneum.
27	18 M	Small cavity left upper lobe ($\frac{3}{4}$″), small fibrotic cavity (1″) right upper lobe.	Pos.	Neg.	Left and right diaphragm $4\frac{1}{2}$″ (no phrenic)	Pneumoperitoneum and right 5-rib thoracoplasty.

* Complication: Pneumocele of umbilicus.

Small sized cavity 0-1″ diameter. Medium sized cavity 1″-2″ diameter. Large sized cavity 2″+diameter.





. 1.—Case No. 3. Radiograph (1). Large ion cavity in left lower lobe with infiltration at periphery of right upper zone.

. 3.—Case No. 9. Radiograph (1). Bilateral se. Cavitation is present in the right infra-icular region and the apex of the right lower . The lesion in the left infra-clavicular region appears to be cavitating.

FIG. 2.—Case No. 3. Radiograph (2). Left phrenic crush and pneumoperitoneum have been performed, with marked elevation of the left hemi-diaphragm and closure of the cavity.

FIG. 4. Case No. 9. Radiograph (2). Right phrenic crush and pneumoperitoneum have been performed with closure of both cavities and regression of the left clavicular lesion.

To face p. 50

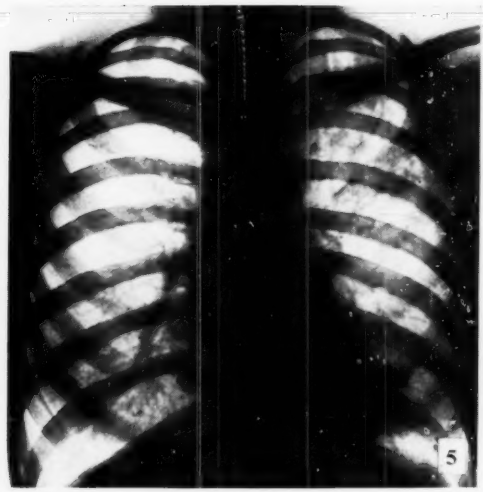


FIG. 5.—Case No. 17. Radiograph (1). A medium-sized tension cavity is situated in the left upper lobe with surrounding infiltration.

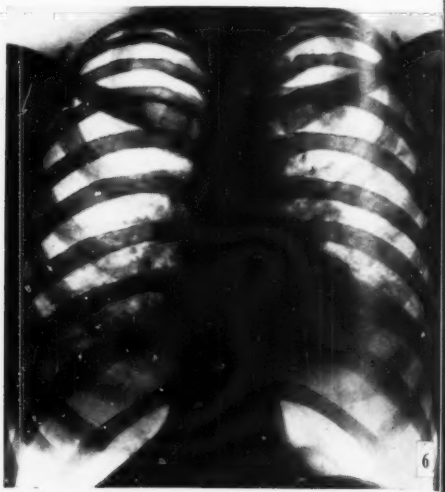


FIG. 6.—Case No. 17. Radiograph (2). Left phrenic crush and pneumoperitoneum have been performed with closure of the cavity and regression of the infiltration.

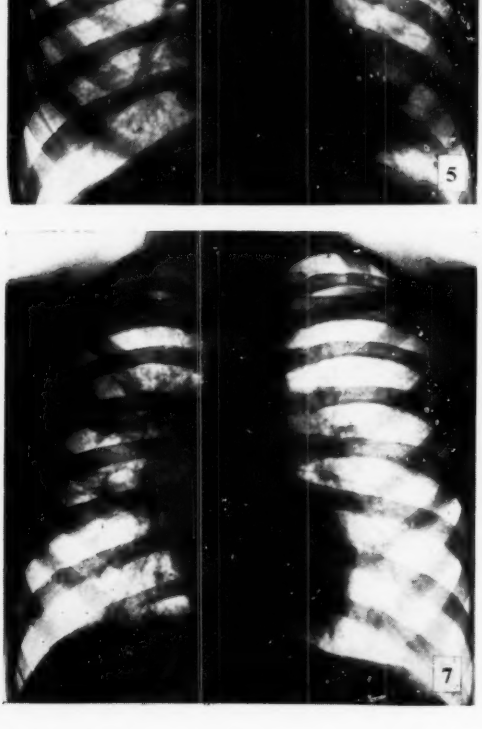


FIG. 7.—Case No. 20. Radiograph (1). A cavity is situated in the upper part of the right lower lobe, and a tension cavity surrounded by infiltration is in the right upper lobe.

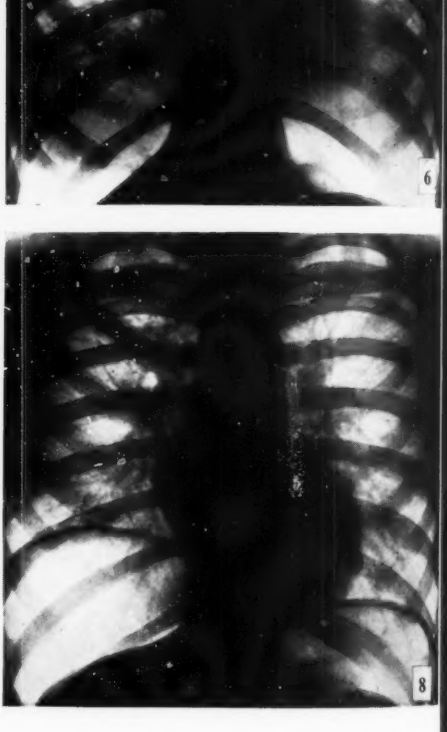


FIG. 8.—Case No. 20. Radiograph (2). Right phrenic crush and pneumoperitoneum have been performed with closure of the cavities and regression of the infiltration.

while in three cases cavitation was present in both upper and lower lobes. Among the unsuccessful cases, six had cavities in the upper lobe, three in the lower lobe, while in four cases cavities were present in both upper and lower lobes.

It is interesting to note that more upper lobe cavities were treated by pneumoperitoneum in the routine admissions, but treatment was relatively less successful, presumably because these were more fibrotic in nature. In this series of routine admissions to my wards at the Yardley Green Hospital, 67·5 per cent. were successfully treated by pneumoperitoneum.

The optimum position for cavity closure was in the apex and upper part of the lower lobe, but the effectiveness of treatment depended also on adequate elevation of the corresponding hemidiaphragm. In those cases where lower lobe cavities were closed, an average elevation of the corresponding hemidiaphragm was 2·7 inches, and for upper lobe cases it was 3·02 inches. Tension cavities reacted favourably under the same conditions. Rapid elevation of the diaphragm by large frequent refills produced maximum disturbance of the broncho-cavitary mechanism and possibly occlusion of the draining bronchi. When bronchogenic disease was present it regressed, providing it was not too confluent.

Consideration of some individual cases will clarify certain significant aspects of this treatment.

In case No. 3 (see illustrations) a giant tension cavity 3 inches in diameter was situated in the middle portion of the posterior part of the left lower lobe. The cavity was closed following an elevation of the left hemidiaphragm by $5\frac{3}{4}$ inches. In the closure of this cavity the following factors played leading parts. With elevation of the diaphragm the cavity walls were brought closer together and also the draining bronchi were occluded. Secretions and caseous material accumulated in the cavity and completely obliterated the lumen. The final picture was that of a " blocked " cavity.

In case No. 9 (see illustrations) three cavities were present. As two of them were situated in the right lung, a right phrenic crush was performed to obtain greater elevation of the right hemidiaphragm and consequent greater reduction in volume of the right lung. This policy is worth while when treating bilateral lesions.

In case No. 17 (see illustrations) the tension cavity situated in the left upper lobe was reduced in size following a left phrenic crush. A pneumoperitoneum produced quick closure of the cavity. This case illustrates how the tension mechanism of a cavity can be upset by the rapid alteration of intra-pulmonary mechanics produced by the recommended scheme of refills.

This effect was also noted in case No. 20 (see illustrations), where two tension cavities surrounded by infiltration were present in the right upper and lower lobes. In addition to rapid closure of the cavities the infiltration regressed markedly.

In thirteen cases pneumoperitoneum was unsuccessful in closing cavities. In eleven, the reason for failure was that the cavity was too fibrotic. Although fibrotic cavities require permanent collapse therapy, it is worth while attempting a short pneumoperitoneum trial when in doubt, as sometimes the exact degree of fibrosis can be misjudged on the radiograph.

UNSUCCESSFUL CASES

Case no.	*Age & Sex*	*Type and position of disease*	*Elevation of diaphragm*	*Reason for failure of pneumoperitoneum*
28	23 F	Medium-sized cavity (1¾″) at periphery of left mid-zone, infiltration right mid-zone	3″	Cavity too fibrotic. Thoracoplasty successful. Infiltration regressed during P.P. treatment.
29	34 M	Large cavity (2¼″) left apex, many small cavities left mid-zone	2½″	Cavity fibrotic at left apex. Diffuse peribronchial fibrosis around mid-zone cavities.
30	19 M	Medium-sized cavity (1¼″) in upper part of right lower zone	2½″	Cavity too fibrotic. Spread occurred to left base.
31	24 F	Medium-sized cavity (1½″) right upper lobe with surrounding dense exudative disease	2½″	Cavity surrounded by soft caseous tissue.
32	11 F	Small cavity (¾″) left upper lobe	2″	Cavity too fibrotic.
33	14 F	Large cavity (2½″) left upper zone, infiltration upper two-thirds left lung. Three small cavities left upper zone with surrounding infiltration	4″	Eight months' bed rest healed infiltration. Resulting fibrosis prevented cavity closure.
34	35 F	Large fibrotic cavity (2½″) right upper zone, recent cavity (1½″) in left upper zone	4½″	Even this elevation did not close left lung cavity—too fibrotic. Thoracoplasty could therefore not be performed on right.
35	35 M	Medium-sized cavity (1½″) upper part of right lower lobe. Calcified foci both lungs	2½″	Cavity too fibrotic.
36	31 F	Medium-sized cavity 1½″ apex right lower lobe	2″	Cavity too fibrotic.
37	20 F	Two medium-sized cavities 1¾″ left upper lobe and apex lower lobe, surrounded by dense exudative disease	2″	Cavities became surrounded by caseous tissue.
38	16 F	Two medium-sized cavities 1¼″ right upper lobe and three small cavities ½″ lower lobe	3″	Diffuse fibrosis surrounding cavities.
39	19 F	Medium-sized cavities 1½″ left lobe and lower lobe	2½″	Cavities were tension type and "ballottement" was unsuccessful. Diffuse fibrosis prevented cavity closure.
40	28 F	Cavity in right upper lobe and apex of lower lobe. Healed infiltration right upper lobe	3″	Diffuse fibrosis in right lung prevented cavity closure.

Small-sized cavity 0-1″ diameter
Medium-sized cavity 1″-2″ diameter
Large-sized cavity 2″ + diameter

In case No. 29 it was evident after a short period that the tension cavity's draining bronchi were surrounded by fibrosis, preventing their permanent closure by pneumoperitoneum. Fibrosis of the cavity wall itself or surrounding tissues plays an important part in preventing closure of the cavity. All efforts should be directed to ascertaining the age of the cavity. The older the cavity the more likelihood there is of its being fibrotic.

In the remaining two unsuccessful cases (Nos. 31 and 37) the cavities were surrounded by dense confluent disease, which became soft caseous tissue. This prevented the full effect of the pneumoperitoneum reaching the cavity.

Discussion

The principal aim of all forms of collapse therapy is the closure of pulmonary cavities, and the true efficiency of each method must be judged by its power to attain this end in the shortest time and with the least disturbance to the patient. Accordingly the only criteria on which its value can be assessed are radiological evidence of closure of the cavity, disappearance of tubercle bacilli from the sputum, and the incidence of complications. An uncomplicated artificial pneumothorax is still recognised as the best means for producing "concentric" collapse, but how often is one able to obtain such perfect conditions? The presence of pleural adhesions not only favours contraselective collapse but is said to increase the incidence of pleural fluid. Attempts to section these adhesions at thoracoscopy increase the liability to further difficulties. If instead of "inducing an artificial pneumothorax" one said "opening the pleural space," one would be placing in true perspective artificial pneumothorax treatment and its attendant difficulties. There is, therefore, a good incentive to find out whether cavity closure can be attained without these risks.

Pneumoperitoneum fulfils to a large degree the requirements postulated above. In this series the small incidence of complications is notable. The maintenance of treatment is easy, and is beset with few difficulties. The ease with which it can be re-induced at a later date, if indicated, further increases its value. Reduction in lung volume can be obtained by pneumoperitoneum without separating the visceral and parietal pleuræ, and so it can be used in the presence of exudative disease with minimal risk of pleural complications.

Study of the cases in this series emphasises the value of pneumoperitoneum as a primary treatment for closing certain types of cavities and as a secondary treatment when it is not possible to induce an artificial pneumothorax owing to pleural symphysis. In selecting cavities for closure by this method, the deciding factors are the age of the cavity and the condition of the surrounding lung. Recent cavities, particularly of small (1 inch or less in diameter) or medium size (1 to 2 inches), situated in any part of the lungs, can be readily closed. The older fibrotic cavities do not respond. It is sometimes difficult to assess the degree of fibrosis, and when in doubt that permanent collapse by thoracoplasty is necessary, a trial of pneumoperitoneum should be conducted for a short period. The surrounding lung tissue may be fibrotic and the full effect of the pneumoperitoneum will not be allowed to reach the cavity. When there is dense exudative disease in the surrounding lung, elevation of the

diaphragm may cause this to coalesce and the cavity becomes the centre of the caseous tissue which prevents it being affected. The optimum cavity position for closure by pneumoperitoneum is in the apex and upper part of the lower lobe. Tension cavities can be closed by pneumoperitoneum. It probably acts by occluding the bronchi draining the cavity. If there is difficulty in closing the tension cavity, " ballottement " should be attempted in the way described. Sometimes even this may be unsuccessful. When there is marked fibrosis around the cavity's draining bronchi, this prevents their being completely and permanently closed. It is important to obtain adequate elevation of the diaphragm when attempting cavity closure; otherwise pneumoperitoneum treatment is not being adequately applied and cannot produce its best effects.

Pneumoperitoneum, without phrenic paralysis, can be used to produce relaxation of both lungs simultaneously. In this way cavities of the above types when situated in both lungs can be treated simultaneously.

This treatment can be used effectively in bringing a patient with bilateral disease within the sphere of surgery. A cavity in one lung can be closed successfully by pneumoperitoneum so that thoracoplasty can be performed on the other lung.

Pneumoperitoneum can produce reduction in lung volume without separation of the visceral and parietal pleuræ. It can be effectively used in the presence of exudative disease where, if an artificial pneumothorax were used, pleural fluid would occur. Pneumoperitoneum has a regressive effect on exudative disease if it is not too confluent.

During the past few years chemotherapy in the form of streptomycin and P.A.S. has been widely available. This has not limited treatment by pneumoperitoneum; on the contrary, it has extended it. Chemotherapy and pneumoperitoneum are not competitive treatments but rather complementary ones. Streptomycin can heal endobronchial tuberculosis and so aid the closure of tension cavities. Many of these have been seen to disappear after this treatment, yet pneumoperitoneum is needed to maintain this state. It has been noted that infiltrations and exudative lesions have regressed during pneumoperitoneum treatment, and if chemotherapy is used in addition, the results can be very satisfactory. Pneumoperitoneum and chemotherapy should be used simulatneously, if either is not contra-indicated, as pneumoperitoneum by closing the cavity will eliminate the danger of bronchogenic spread always present with a patent cavity.

The present bed shortage for tuberculous patients emphasises the importance of pneumoperitoneum. Its minimal complications and ease of management allow it to be used as a successful domiciliary treatment if home conditions are satisfactory. Best use can be made of limited beds by using them for " short-term cases." A pneumoperitoneum is induced and phrenic crush performed during a hospital stay of a few weeks. The patient is then discharged home to rest in bed and attend the Chest Clinic by ambulance for refills. This procedure may be preceded by chemotherapy at home while waiting a vacancy. In this way, maximum use can be made of minimum beds.

Personal experience with pneumoperitoneum leads the writer to conclude that it is a valuable, efficient and safe method of cavity closure and so plays an important rôle in the modern treatment of pulmonary tuberculosis.

Summary

The literature on pneumoperitoneum in the treatment of pulmonary tuberculosis is briefly reviewed and various aspects of the treatment, with reference to personal experience, are discussed.

The indications for the use of pneumoperitoneum in closing cavities are defined and suggestions made for its extended use.

A personal technique and management of this treatment are elaborated and increased use of this method is suggested.

This paper is based on part of a thesis accepted for the degree of M.D. (Wales).

I wish to thank Dr. J. E. Geddes, Senior Consultant Chest Physician, City of Birmingham, and Mr. A. L. d'Abreu, Consultant Thoracic Surgeon to the Yardley Green Hospital, for their encouragement in the pursuit of this subject.

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SPONTANEOUS PNEUMOTHORAX COMPLICATING PNEUMOPERITONEUM THERAPY

By J. H. P. Johnson

From the East Ham Chest Clinic, London

Spontaneous pneumothorax occurring as a complication of pneumoperitoneum treatment is one of the rarer hazards of this form of therapy. Occasional cases have, however, been reported since Mellies' (1939) first recorded case, and that the incidence is higher than the literature indicates is shown by the fact that Ross and Farber (1951) discovered twelve cases additional to their three by submitting a questionnaire to a group of sixty-one chest physicians in California.

Twenty-four cases have been reported in the literature and in each case the pneumothorax has been on the right side. Smith (1943) reported a fatal case of bilateral spontaneous pneumothorax following the induction of a pneumoperitoneum, in which autopsy revealed several small holes in both halves of the diaphragm; this is the only instance of a left-sided pneumothorax. The only other fatal case was described by Yannitelli *et al.* (1949), and autopsy showed what appears to have been a traumatic rupture of the right dome of the diaphragm. In no other case is there any direct evidence as to the path which the air has taken in passing from the abdomen into the thorax, although the supportive evidence in some cases is convincing.

A further case is now presented in which air from a therapeutic pneumoperitoneum entered the right pleural space following trauma.

Case Reports

G.C., a woman aged 59 years, came under treatment in February 1952, when, following a small hæmoptysis, she was found to be suffering from pulmonary tuberculosis with infiltration in both upper zones and a left apical cavity; her sputum contained acid-fast bacilli, her E.S.R. was 6 mm. in one hour (Westergren), and her Vital Capacity was 1,800 c.c.

She was admitted to the Plaistow Hospital Chest Unit under the care of Dr. Philip Ellman on 2.4.52 and remained there till 11.8.52. During this period she was kept at rest and given a course of streptomycin 1 G. intramuscularly daily and P.A.S. 20 G. by mouth daily for 130 days. She improved on this régime, but as the cavity, though smaller, was still patent and her sputum was still positive, a pneumoperitoneum was induced on 10.6.52. This resulted in a satisfactory elevation of the diaphragm, and after a few weeks her sputum became negative and the cavity could not be seen even on tomography. It was noticed that the diaphragm was unusually thin, but this was not considered to contra-indicate continuing the pneumoperitoneum.

Following her discharge from hospital the pneumoperitoneum was maintained without difficulty and, as her disease continued to remain quiescent, she was allowed up for increasing periods till she was up all day.

On 6.3.53, while going to the Clinic for her refill, she fell heavily on her

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FIG. 1.—Skiagram showing the pneumoperitoneum to be well established after the first accident and before the second.

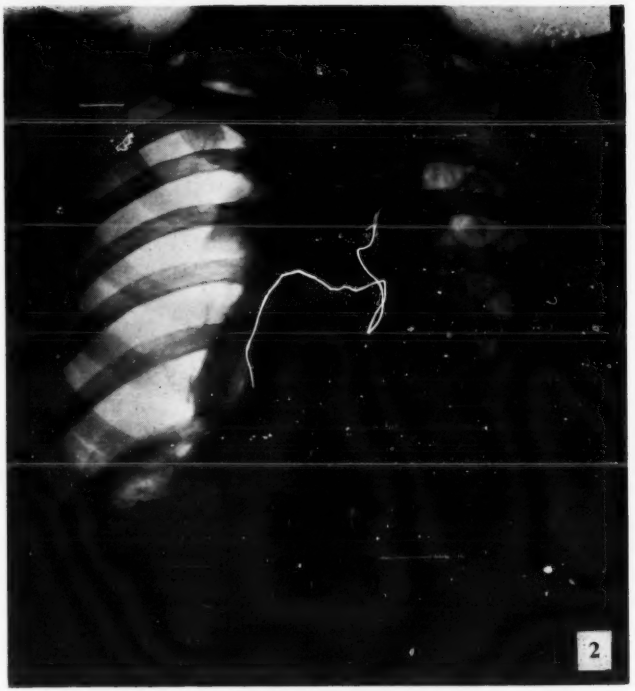

FIG. 2.—Skiagram after the second accident, showing disappearance of the pneumoperitoneum and a right spontaneous pneumothorax.

left shoulder; the pneumoperitoneum appeared as usual on radioscopy and she was given her customary refill of 1,000 c.c., the pressures being +9+10 before refill and +10+11 after refill (Fig. 5). Throughout the next week she had a feeling of heaviness in her chest and, at the end of this period, while lying in bed this sensation increased abruptly, she was breathless and had to sleep propped up that night. The following morning she felt better, but when she arrived for her refill she complained of a stifling sensation. Radioscopy now disclosed a right spontaneous pneumothorax and the pneumoperitoneum had almost disappeared; she was not refilled then but sent home to rest.

On 27.3.53 a shallow pneumothorax was still present but the pneumoperitoneum was no longer visible. Refill needles connected to two separate pneumothorax apparatuses were introduced simultaneously into the right thorax and abdomen, and the pressures were found to be -7 ± 0 in the chest and +10 in the abdomen. After filling 500 c.c. of air into the peritoneal cavity the pressures in both chest and abdomen were unaltered. She then sat up without discomfort, and the pressure in the abdomen rose to +20 while that in the chest remained unaltered. She was then replaced in the supine position and a further refill of 500 c.c. was given, the final pressures being +9+10 in the abdomen and -7 ± 0 in the right chest. Radioscopy now showed the pneumoperitoneum again to be satisfactory, the diaphragm having risen well and moving normally; the right pneumothorax remained as shallow as previously.

In the next fortnight the pneumothorax completely reabsorbed and the pneumoperitoneum was satisfactorily maintained till 31.5.53, when, while seated in a chair, she made a sudden turning movement and again experienced a feeling of tightness in her chest (Figs. I and II). Radioscopy the following day showed a recurrence of the right spontaneous pneumothorax and the pneumoperitoneum was again shallow. As the right lung was almost completely collapsed 700 c.c. of air was withdrawn from the pleural space, the pressures before and after being -3 +3 and -4 -1.

Thoracoscopy on 10.6.53 showed "A trace of turbid fluid in the pleural space but no gross pleural disease; there were a few divisible apical adhesions. No abnormality was seen in the diaphragm, but a large area was obscured by adhesions to the lung surface."

No more pneumoperitoneum refills were given and the air in the pleural and peritoneal spaces reabsorbed in the next month. This series of episodes had no appreciable effect on the pulmonary disease, which remained quiescent throughout; the patient remains well and asymptomatic at the present time.

Discussion

From the scanty autopsy material it is clear that a congenital defect or a traumatic tear of the diaphragm are possible causes of such pneumothoraces, and the evidence suggests that the air usually passes directly through the diaphragm rather than by a devious route. The theory has been advanced by Banyai and Jurgens (1940), Simmonds (1946) and Loughead (1950) that the air tracks through the aortic or œsophageal hiatuses of the diaphragm into the mediastinum and then ruptures into the pleural space. There is little to support this theory, which would postulate a defect in the parietal peritoneum at the hiatus and a defect in the pleura in another area. Furthermore, there is no evidence that mediastinal emphysema occurred in any of the cases, and the theory does not explain why no pneumothoraces have occurred on the

left side. This is contrary to what one would expect, as Dickie (1948) has shown that, when pneumothorax complicates mediastinal emphysema, the incidence is equal on both sides.

That some cases are due to congenital defect in the diaphragm is further supported by the case described by Sita-Lumsden (1949), in which a patent pleuro-peritoneal canal was probably present. He argued that such defects, if confined to the right side, would be protected by the liver and only become apparent when the diaphragm loses this protection on inducing a pneumoperitoneum. The explanation, which accounts for the preponderance of the spontaneous pneumothoraces on the right side as being due to the fact that diaphragmatic defects on the left side usually become manifest and preclude the use of pneumoperitoneum, does not appear to be wholly correct. There can be little doubt that the true incidence of developmental diaphragmatic defects is, in fact, predominantly left-sided, and this is explained by the earlier closure of the pleuro-peritoneal opening on the right side. That latent defects of the left diaphragm do occur is shown by the fact that, of three cases reported in the literature (Banyai, 1933; Anderson and Grenville-Mathers, 1951) where a spontaneous pneumoperitoneum occurred as a complication of artificial pneumothorax, the latter were in two instances on the left side.

It seems probable, as pointed out by Spencer Jones and Yuill (1952), that two types of case exist—those which follow closely on the induction of a pneumoperitoneum, due to a congenital defect in the diaphragm, and those in which the pneumothorax follows some time later, due to trauma. The latter appear to be the commoner and occur where there is a weakness in the diaphragm together with a sudden rise of intra-abdominal pressure. Such weakness may be due to a general hypoplasia or a localised defect due to thinning or separation of a few fibres, or to an inflammatory lesion as in Mellies' case (1939). In several cases blebs or bullæ have been noticed above the diaphragm either radiologically or through the thoracoscope. In Repa and Jacobson's case (1951) the bleb disappeared when the spontaneous pneumothorax occurred, and in Cope's case, reported by Ross and Farber (1941), thoracoscopy revealed a large bleb which varied in size with respiration. Laird (1945) reported similar blebs in four patients who were receiving combined pneumoperitoneum and pneumothorax treatment. It is generally agreed that the presence of phrenic paralysis plays little part in the incidence of pneumothorax in cases with pneumoperitoneum. Phrenic paralysis was absent in the case now described, and thinning of the diaphragm was apparent radiologically immediately the pneumoperitoneum was induced and before any significant stretching could have occurred. A congenital hypoplasia seems probable and rupture was caused by indirect trauma. Harrington (1948) pointed out that diaphragmatic hernias following indirect trauma are predisposed to by congenital inadequacies of the diaphragm and usually occur at the line of fusion of its embryological elements; this would also seem to apply to the type of case under discussion.

Apart from the autopsy case reported by Yannitelli *et al.* (1949), probable tears due to indirect trauma were seen through the thoracoscope in the cases described by Wynn-Williams (1950), Repa and Jacobson (1951), and Spencer Jones and Yuill (1952). In the present case thoracoscopy was done three

months after the first incident, and the adhesions between the lung and diaphragm may have resulted from a tear, although a previous fibrinous pleurisy is equally possible. In a third of the described cases there is a history that the accident was preceded by indirect trauma in the form of abdominal strain, sudden movement or a fall, all of which are likely to cause a sudden rise of intra-abdominal pressure. It is interesting to speculate whether in such cases excessive pressure could be exerted on the right diaphragm owing to a piston-like action of the liver moving upwards while the abdominal muscles are tense. The recurrence of the pneumothorax some weeks later in this and other cases suggests a traumatic rupture rather than a developmental hiatus in the diaphragm.

Conclusions

It is clear that for this complication to occur there must be a diaphragmatic defect. If there is a patent developmental hiatus, the air passes at once into the pleural space when a pneumoperitoneum is induced; if, on the other hand, there is weakness of part or the whole of the diaphragm, or if the elements from which it is developed are poorly fused, rupture would more likely follow a sudden rise of intra-abdominal pressure. In such cases a pocket of high pressure under the right diaphragm, or the unmasking of a latent defect when the support of the liver is lost on inducing a pneumoperitoneum, may account for the predominance of such lesions on the right side.

Summary

Spontaneous pneumothorax due to air from a pneumoperitoneum is shown to be a well-recognised complication of this treatment and a further case is described.

Traumatic rupture of a weak diaphragm appears to account for the majority of the cases, although developmental defects are occasionally responsible.

An explanation is offered for the preponderance of the pneumothoraces on the right side.

I would like to thank Dr. Philip Ellman for his permission to publish this case and for his helpful criticisms. I would also like to thank Mr. K. Shaw for the thoracoscopy report.

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"LIPIODOL SWALLOW" AND PARAFFINOMA OF THE LUNG

By Lucy M. B. Nelson

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Much has been written on lipoid pneumonia and paraffinoma in man and the changes which follow the instillation of oils in experimental animals, but stress is laid on these conditions as afflictions of the helpless, the aged or the infirm, and unrelated to vigorous men in the prime of life unless an accident occurs, such as the bursting of an oil pipe, allowing massive oil inhalation. Because of the danger to the lower respiratory tract nasal drops are seldom now made up in paraffin, but there may be many adults who regulate habitual constipation with liquid paraffin at night, and while traces still remain in the pharynx fall deeply asleep or ensure sleep with hypnotics. As they sleep, droplets of paraffin are inhaled and lodge in the dependent part of the lung.

When Ikeda (1937) described his own findings in cases of lipoid pneumonia and reviewed previously reported cases, he divided these into infantile and adult types and stressed that this granulomatous foreign body reaction was more common than had hitherto been suspected and that paraffinoma should always be borne in mind in the differential diagnosis of obscure pulmonary conditions.

As early as 1920, Guieysse-Pelissier had described the lung changes of lipoid pneumonia, and in 1925 Laughlin drew attention to the death of three children following the instillation of oil into the nose and of the death of an adult with dysphagia. The adult had been taking 1½ ounces of liquid paraffin three times daily by mouth during the last four and a half months of his life. Graef (1939) stressed the dangers of intra-nasal medication with oil and advised that care should be taken to prevent aspiration when oil was given orally.

Engleberg, Frieman and Merrit (1940), Pinkerton (1940), Janes (1947) and other writers have described numerous cases occurring in babies, the debilitated and the dysphagic to whom liquid paraffin had been given, but it has also been recognised that in the adult type there is by no means always interference with swallowing. Berg and Burford (1950) and Janes (1947) suggested individual susceptibility as a possible factor.

The history may not only suggest the diagnosis but may indicate the distribution of the lesion. Brown and Biskind (1941) postulated that the rapid inhalation of large amounts led to diffuse, and frequent small aspirations to localised, lesions. There have been occasional puzzling cases (Rewell, 1947, where there was no history of oil administration to help in the diagnosis) and it may even have been missed at the post-mortem examination.

Further help in the differential diagnosis may be suggested by the radiological changes (Moel and Taylor, 1943; Davis, 1936). The sputum may show

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free oil (Bishop, 1940), or oil containing secretions may be bronchoscopically obtained (Singer and Tragerman, 1941). A further clue may be given if the patient's sleep posture is known, as in Stokes's (1943) case who slept on the right and developed a lesion in the right middle and lower lobes. In some cases needling of the lung (Thelwall Jones, 1952) has provided the diagnosis.

The danger of delay in diagnosis is increased by possible malignant change, as in Wood's (1943) case where bilateral carcinoma of the lung was associated with lipoid pneumonia. A similar warning is given by Berg and Burford (1950).

Case History

H.M., a man of 46, was admitted to Broadgreen Hospital on 10.6.52 with a history of recurrent slight hæmoptysis for four years and occasional pain in the left chest, sometimes pleuritic in type. There was no loss of weight and his general condition was good. He was a non-smoker. He was X-rayed on 11.6.52 (Fig. 1) and the film showed an abscess cavity in the basal segments of the left lower lobe.

12.6.52 (Fig. 2). Left bronchogram showed obstruction in the region of the anterior basal and lateral basal segments of the left lower lobe and cylindrical bronchiectasis of the lingula. 19.6.52: a right bronchogram was normal.

1.7.52. At thoracotomy a hard craggy mass involving the anterior basal and lateral basal segments was found and a left lower lobectomy was carried out by Mr. F. R. Edwards. The lobe was examined by Dr. Whitwell, who reported on 21.7.52 that stained sections showed the presence of mineral oil, the estimated amount of oil in the lobe being about 16 grammes.

Here was a vigorous man who as chief engineer supervised the work of others and had no direct contact with mineral oil at work. He had, however, for years been in the habit of taking ½ ounce of liquid paraffin at 9 p.m. and for at least two years had followed this with a barbiturate, retired to bed at 10 p.m., and for the first few hours lain face downwards.

Mr. McFarland was asked to examine his larynx in case there was some abnormality in swallowing. He found no evidence of abnormality in the pharynx, cords or larynx.

It was decided to screen him in the act of swallowing a radio opaque oil; this was done on 4.10.52 (Fig. 3). A small amount of lipiodol was seen to pass partly down the œsophagus and partly down the trachea without evoking a cough reflex. This happened each time he swallowed the oil, so that it was possible to take films. A P.A. film of his chest immediately afterwards showed traces of lipiodol in the left lower chest (Fig. 4). He had demonstrated quite simply how the oil had accumulated in the previous months. As soon as the liquid paraffin became suspect he stopped taking it, and a year after his original admission a lipiodol swallow was again carried out. This time it took some manœuvring to persuade the oil down the larynx; that is, sometimes none went down but if he threw his head back as he swallowed there was a trickle over the larynx (Fig. 5). A series of seven normal people have been similarly screened during lipiodol swallowing; in all of them the lipiodol shoots swiftly and straightly down the œsophagus (Fig. 6), as does barium in this particular patient.

It may be that this man has always had some defect in swallowing; if so it is perhaps strange that his lungs should have suffered only during that four-year period. Again, it may be that the not uncommon practice of taking mineral oil at night and inducing quick sleep with barbiturates is a dangerous combination which may lead to repeated small aspirations in otherwise healthy people.

Method. One drachm of lipiodol was held in the mouth and swallowed as little as possible at a time till none remained in the mouth. Lipiodol contains 39 to 41 per cent. w/w of combined iodine: no toxic effects have been noticed in the dosage used and peppermint sweets have counteracted the slight oily after-taste.

Summary

A case of paraffinoma of the lung in an otherwise healthy man who for years combined a nightly dose of liquid paraffin with barbiturates is described.

If clinically or radiologically paraffinoma is suspected, screening the patient while he swallows lipiodol may point to a diagnosis before the chest is opened.

I would like to thank Dr. Whitwell for stimulating my interest in the case, Mr. Edwards for allowing me to see and follow-up the case, and Dr. Rubin and Mr. Wall for all their help in the radiological part of the investigation.

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FIG. 1.—June 11, 1952. P.A. X-ray of chest showing abscess cavity in left lower lobe.

FIG. 2.—June 12, 1952. Lateral view after left bronchogram showing obstruction of the anterior and middle segmental bronchi and some dilatation of the lingular bronchus.

FIG. 3.—October 4, 1952. The larynx outlined while swallowing lipiodol.

FIG. 4.—October 4, 1952. Lipiodol in bronchi along left border of heart from 2nd to 3rd intercostal spaces immediately after swallowing.

FIG. 5.—June 20, 1953. Larynx outlined while swallowing lipiodol; there is some barium in the œsophagus.

FIG. 6.—Two normal controls swallowing lipiodol: none enters the larynx.

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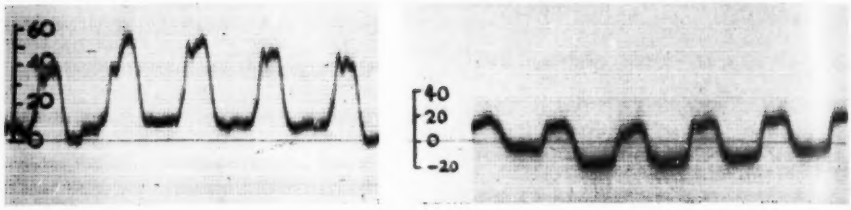

FIG. 1.—Right ventricular pressure tracings showing a reduction of resting pressure with recovery from congestive cardiac failure. (A) In failure the pressure was 43/8 mm. Hg; (B) Two weeks later during recovery from failure it was 19/-5 mm. Hg.

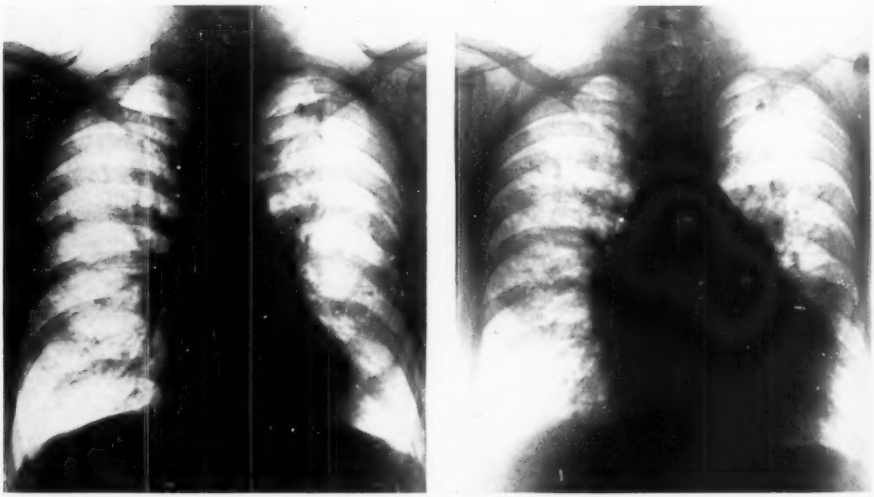

FIG. 6.—Teleradiogram of chest. (A) Patient ambulant; no congestive phenomena. (B) Patient in gross heart failure during an attack of acute bronchitis. Both are 2-metre, P-A films. With recovery the heart size was again comparable with (A).

EMPHYSEMA HEART DISEASE

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Our understanding of the functional pathology of emphysema heart disease has recently been expanded by new methods of investigation of the respiratory and cardiovascular systems. Studies by cardiac catheterisation have disclosed the largely episodic nature of pulmonary hypertension in this disease and have emphasised the importance of energetic treatment of the exacerbations of bronchitis which seem to precipitate heart failure.

Incidence

The incidence of emphysema heart disease both in relation to emphysema and in relation to other diseases is difficult to assess. This is partly due to the fact that patients with this disease previously tended to congregate in municipal hospitals and were therefore rare in teaching hospitals. Series of cases gathered from these two different sources would tend to give a very different picture of the incidence of the disease. Parkinson and Hoyle (1937) found evidence of right ventricular hypertrophy on radiological examination in only 18 out of 80 cases of emphysema. On the other hand, in an autopsy series of 74 cases of emphysema at Hammersmith Hospital, 57 per cent. were found to have evidence of right ventricular hypertrophy (Lewes, personal communication). White and Jones (1928) saw only 21 cases of emphysema heart disease in 2,314 cases of organic heart disease. At Hammersmith Hospital, on the other hand, the incidence of emphysema heart disease at autopsy was considerably higher. Of a total of 3,495 consecutive autopsies over a ten-year period, 128 cases of right ventricular hypertrophy due to chronic bronchitis and emphysema were seen. The relative frequency of emphysema heart disease at this hospital can be appreciated from the fact that the incidence at autopsy of left ventricular enlargement due to hypertension over the same ten-year period was only four and a half times as great as that of right ventricular hypertrophy due to emphysema (Lewes, personal communication).

Chronic bronchitis is largely a disease of middle-aged men (McMichael, 1948; Oswald *et al.*, 1953), and hence heart disease due to chronic bronchitis and emphysema shows a similar sex and age incidence.

Pathogenesis

Pulmonary Hypertension.—Fixed structural changes in the pulmonary vascular bed, which increase its rigidity or reduce its cross-sectional area, have previously been considered to play the major part in the production of high

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pulmonary arterial pressures in emphysema. Recently, however, by means of cardiac catheterisation studies, the high resting pressures recorded during cardiac failure have been shown to be transitory and reversible (Figs. 1 and 2)

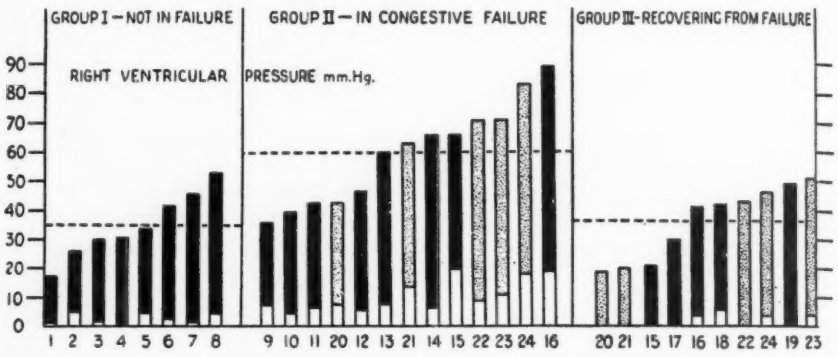

FIG. 2.—Right ventricular pressures of twenty-four patients at rest. Peak systolic pressure is indicated by the shaded portion of the bar; end-diastolic pressure, by the clear portion of the bar. Mean values for the three groups are shown by the horizontal broken lines. Stippled bars represent those patients who were studied twice during a single hospital admission for congestive cardiac failure.

(Ferrer *et al.*, 1950; Mounsey *et al.*, 1952). Permanent structural changes, therefore, cannot alone be responsible for the transient high pressure, although they almost certainly play some rôle in maintaining the moderate pulmonary hypertension seen at rest in patients not in congestive cardiac failure (Borden *et al.*, 1950; Harvey *et al.*, 1951; Mounsey *et al.*, 1952). There is no correlation, however, between residual air percentage, as indicating the degree of emphysema, and the right ventricular pressure in severely emphysematous patients (Harvey *et al.*, 1951; Mounsey *et al.*, 1952). Pulmonary hypertension may also result from acute bronchitis alone in the absence of reduction of the pulmonary capillary bed from alveolar destruction (Clinico-Pathological Conference, 1951).

Certain functional factors must therefore be considered in the mechanism of production of pulmonary hypertension in chronic bronchitis and emphysema. The arterial saturation with oxygen is decreased when patients are in congestive cardiac failure and have raised right ventricular pressures, but, with recovery from failure, the saturation returns to a higher level as the pressure falls. This association between a fall in pressure and a rise in arterial saturation is most clearly seen in individual patients (Fig. 3), but a similar correlation is also seen in a series of patients considered collectively (Fig. 4) (Borden *et al.*, 1950; Harvey *et al.*, 1951; Mounsey *et al.*, 1952). Correction of anoxia, however, during congestive cardiac failure with oxygen treatment does not always produce an immediate fall in pressure (Mounsey *et al.*, 1952). A possible complicating factor may be the associated increase in arterial CO_2 tension known to occur in emphysematous patients breathing high oxygen mixtures (Taquini *et al.*, 1948).

A less direct relationship of anoxia to pulmonary hypertension may be postulated through its effect on blood volume. Hurtado *et al.* (1945) have

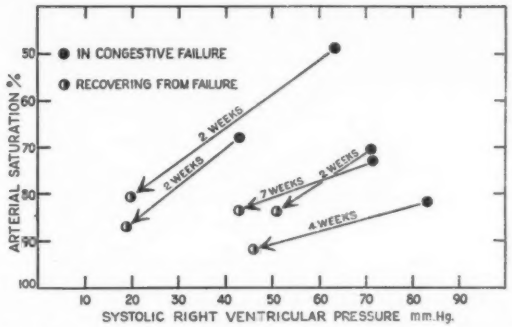

FIG. 3.—Changes in right ventricular systolic pressure and arterial oxygen saturation at rest in five patients during recovery from congestive cardiac failure.

shown that chronic anoxia may cause an increase in blood volume, predominantly increasing the red cell mass. Ferrer *et al.* (1950) and Harvey *et al.* (1951) have shown that in chronic cor pulmonale in failure, the red cell volume increases out of proportion to the plasma volume. Since a fall in blood volume usually occurs on recovery from heart failure, it is quite possible that this reduction in engorgement of the circulation may be one of the factors contributing to the relief of pulmonary hypertension.

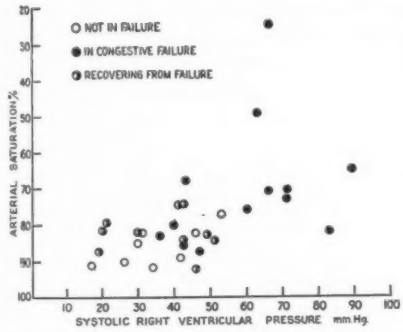

FIG. 4.—Relationship between the arterial oxygen saturation and the right ventricular systolic pressure at rest ($r=0{\cdot}516$; $P=0{\cdot}01$).

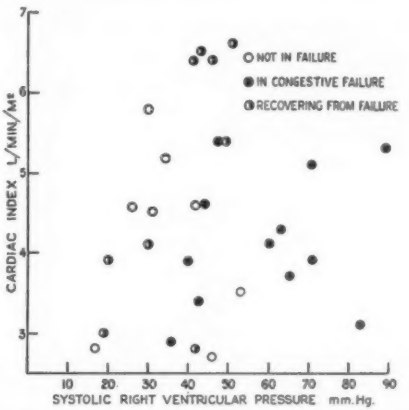

FIG. 5.—Relationship between the cardiac index and the systolic right ventricular pressure ($r=0{\cdot}11$).

Alterations in the pulmonary blood flow in emphysema may also influence the height of the pulmonary vascular pressure. The pulmonary arterial pressure rises during exercise as the cardiac output increases. In normal subjects, on the other hand, little or no alteration in pulmonary arterial pressure results from changes in blood flow (Hickam and Cargill, 1948; Riley *et al.*, 1948). The abnormal response to an increased flow in emphysematous patients during exercise has been explained as resulting both from

restricted capacity and limited distensibility of the pulmonary vascular bed. At rest, however, it is unlikely that levels of flow are high enough to be directly responsible for the production of pulmonary hypertension, and Mounsey *et al.* (1952) found no relationship between the height of the right ventricular pressure and the cardiac index (Fig. 5).

The extent to which neurogenic activity influences circulation through the pulmonary vascular bed remains uncertain, but recently Gilmore *et al.* (1952) have presented evidence suggesting that it could play an important rôle.

Cardiac Output

McMichael and Sharpey-Schafer (1944) showed that normal or even increased cardiac output is present in heart failure secondary to lung disease. This has since been considered as one of the essential features distinguishing it from left ventricular failure. Mounsey *et al.* (1952) confirmed these observations, emphasising the fact, however, that the arterio-venous difference usually remains normal in emphysema heart disease and that the cardiac output is therefore appropriate to the raised level of oxygen consumption present in these breathless patients.

Clinical Picture

The clinical diagnosis of the onset of cardiac complications in chronic bronchitis and emphysema is a matter of considerable difficulty, since symptoms due to emphysema heart disease are gradually superimposed upon those of pulmonary emphysema.

Three major patterns of onset of pulmonary symptoms are met (Mounsey *et al.*, 1952). There are those who have been subject to repeated episodes of winter colds and " bronchitis," which gradually increase in severity and begin to recur each year. Some date their symptoms from a specific respiratory illness, usually described as " pneumonia." Others give a long history of asthma with the gradual development of a persistent cough. Regardless of the type of onset, the symptoms become similar in all patients with increasing severity of their pulmonary disease and the gradual development of cardiac complications. They have a round-the-year cough, usually productive of sputum, and increasing dyspnœa on exertion. Their exercise tolerance varies considerably: cold weather, fog and minor respiratory infections all increase their dyspnœa; patients subject to asthma, however, may be most incapacitated in the summer. Owing to interference with the normal oxygenation of the blood, they gradually become cyanosed. As the disease progresses, recurrent acute exacerbations of bronchitis incapacitate them more, until each exacerbation is attended by an attack of severe anoxia and frank congestive cardiac failure. Many patients die within a year of the onset of failure.

Physical signs of pulmonary hypertension may be partly obscured by those of emphysema. Owing to the anatomical changes in the thoracic cage usually associated with emphysema, clinical estimation of the cardiac size is usually impossible. For the same reason heart sounds are faint over the precordium and an accentuated pulmonary second sound is heard only rarely. Forceful pulsation in the epigastrium, suggesting right ventricular hypertrophy, is often

present. By auscultation in the epigastrium it is sometimes easier to detect the triple rhythm of failure than over the precordium. The presence of a palpable liver should not be considered a sign of venous congestion, since most of these patients have a low diaphragm.

The general clinical picture of patients with emphysema heart disease is well known. They have a dusky blue cyanosis and in some cases mild curvature of the finger nails is present. Obvious clubbing usually signifies more pulmonary pathology than mere chronic bronchitis—*e.g.*, bronchiectases or fibroid changes. Their conversation is often punctuated by laboured breathing. Their thoracic cage is barrel-shaped and its movements limited. On auscultation of the chest, there may be signs of bronchial obstruction with widespread râles and rhonchi, and expiration is always prolonged. In attacks of congestive cardiac failure, there is marked cyanosis, the neck veins are distended and there is often considerable œdema. In cases with severe anoxia there may be drowsiness and delirium. Papillœdema may sometimes be present, without localising signs of intracranial disease.

X-rays of the chest may show right ventricular and pulmonary artery enlargement in addition to bronchitis and emphysema, but are probably not helpful in the early diagnosis of cardiac complications in emphysema. Cardiac enlargement is frequently seen in the presence of congestive cardiac failure (Fig. 6). Similarly the development of cardiographic evidence of right ventricular hypertrophy is often a relatively late event in the clinical course of emphysema heart disease and is seldom of help in diagnosing the inception of the disease. Lead V_4R, on the right side of the chest, was found to be useful in this respect, since the development of an RSR^1 pattern in this lead was sometimes the earliest electrocardiographic sign of right ventricular hypertrophy (Fig. 7) (Mounsey *et al.*, 1952). The hæmoglobin level may be raised, but this is not a constant finding.

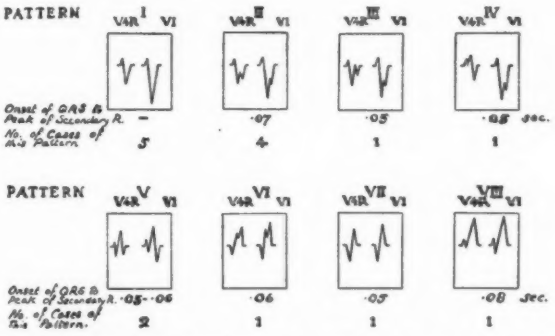

FIG. 7.—Diagram illustrating QRS patterns in VI and V_4R seen in sixteen cases of severe pulmonary emphysema. Patterns I and II show no evidence of right ventricular hypertrophy. In patterns III and IV an RSR^1 complex in V_4R gives evidence of right ventricular hypertrophy. In patterns V to VIII both V1 and V_4R show evidence of right ventricular hypertrophy.

TREATMENT

Although no treatment is available to avert the steadily progressive downhill course of emphysema heart disease, much can be done to help the patient

in attacks of congestive heart failure. Mounsey *et al.* (1952) noted that episodic pulmonary hypertension in heart failure was often associated with acute exacerbations of bronchitis, and it is probable that acute or subacute respiratory infection plays an important rôle in the production of pulmonary hypertension. In the treatment of congestive heart failure, therefore, antibiotics, selected according to the flora in the sputum, should always be given. Treatment with bronchodilators is also useful. Oxygen therapy is of great value in combating hypoxia, but is not without danger, since it may induce carbon dioxide narcosis.

The response to mercurial diuretics during episodes of congestive failure is often good. A low salt diet may be helpful. The value of digitalis in the treatment of emphysema heart disease is probably not as great as in other forms of heart failure, but studies by cardiac catheterisation show that in the acute experiment a higher right ventricular pulse pressure is maintained at a lower diastolic filling pressure and this probably indicates improvement of myocardial function (Ferrer *et al.*, 1950; Mounsey *et al.*, 1952).

Summary

A brief account is given of the way in which studies by cardiac catheterisation have added to our understanding of the pathogenesis and treatment of emphysema heart disease.

I am grateful to Professor McMichael for his help in the preparation of this paper and to the editor and publisher of the *British Heart Journal* for permission to reproduce the figures.

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STREPTOMYCIN AND THE FŒTUS

By A. Sakula

From the Kingston Chest Clinic, Surrey

It is an interesting fact that streptomycin has not frequently been seen to be toxic to the human fœtus, especially in view of the work of Molitor *et al.* (1946), who demonstrated that streptomycin given to weanling rats led to a retardation of growth and the development of pronounced nervous excitability. As this was not noted with adult rats it was suggested that the immature organism might be more susceptible than the adult to the neurotoxic effects of streptomycin.

Experiments on guinea-pigs (Riskaer *et al.*, 1952) showed that streptomycin and dihydrostreptomycin passed from the mother to the fœtus, in, however, a very low dilution but sufficient to be toxic to the fœtus, often leading to its death, and abortion. Those fœtuses which survived manifested no harmful effects on the eighth cranial nerve functions. The drugs were also found to produce toxic effects on the placenta, in which there was hyperæmia and hæmorrhage and commencement of endometrial necrobiosis. These workers suggested that streptomycin given to women early in pregnancy might act as an abortefacient.

In women also, streptomycin traverses the placenta and is readily detected in the fœtal circulation, as has been shown by Heilman *et al.* (1945). Varying amounts of streptomycin were administered to pregnant women at varying times prior to delivery, and simultaneous specimens of blood from the mother and the umbilical cord showed that the concentration of streptomycin in the fœtal blood varied according to the dose and the time between the last injection and birth.

Similar experiments by Woltz and Wiley (1945) confirmed these findings. One injection of streptomycin was given intravenously to fourteen women in labour, and the amniotic fluid and cord blood were assayed and found to contain generally less than one-half the concentration of streptomycin in the maternal blood, but still in an effective antibiotic level. Grasset *et al.* (1951) performed a similar investigation on thirty women, but at different stages of pregnancy, and found the fœtal concentration of streptomycin to be 2·5 to 10 times less than that of the mothers. Levin *et al.* (1948), using dihydrostreptomycin in experiments on three women, also found the cord blood concentration much less than that of the maternal blood.

The first clinical report of the use of streptomycin in pregnancy and its effect on the fœtus, was by Watson and Stow (1948), who administered streptomycin to two tuberculous pregnant women in the second and third trimesters. One had pulmonary tuberculosis and received 90 gm., and the other had urinary tract tuberculosis and received 95 gm., both in 2 gm.

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daily dosages. Both mothers developed some loss of vestibular function, but there were no placental changes. Both the infants were perfectly normal, and a follow-up of one year showed them to have no evidence of involvement of the acoustic or vestibular functions of the eighth cranial nerve. The authors however felt that one should hesitate to give streptomycin in the first trimester, because of the generally accepted sensitivity of fœtal structures in the anlage stage during that period.

There were subsequently further reports by Sala and Laplace (1949), Kistner (1950), Bernard *et al.* (1950), Rogers *et al.* (1951), Froman (1951), Faas (1951), Guzman *et al.* (1952) and Geeraert (1952). All these authors confirmed that streptomycin was innocuous to the fœtus, using the 1 gm. daily dosage. Schaich (1952), however, still urged caution in the use of streptomycin in pregnancy.

Very careful tests of hearing and vestibular function (including a modified Baranyi technique) were carried out on infants by Rubin *et al.* (1951), and they did not find that streptomycin had any toxic effect on the eighth cranial nerve.

A search of the literature has revealed only one reference to any harmful effect to the fœtal eighth cranial nerve by streptomycin. Leroux (1950) described the case of a mother with phthisis who was given only 30 gm. streptomycin, in 1 gm. daily dosage, during the last month of her pregnancy. The baby at 2½ months was quite deaf, although as far as could be ascertained the vestibular function and balance were normal. However, even in this case, there was no definite proof that the congenital deafness was due to streptomycin.

By way of illustration of the clinical problems involved, there follows a description of a woman who received 90 gm. of streptomycin commencing at the tenth week of her pregnancy, with benefit to herself and no harmful effect on the fœtus.

Illustrative Case Report

The patient was a married woman, aged 23, who had been quite healthy until her first confinement two years previously. Following this she had lost weight and in recent months she had been lacking in energy and had developed a slight cough. Five days prior to her first being seen, she had coughed up a small quantity of blood and had developed some pain in the right chest.

On examination, she was found to be in only fair general condition, toxic (temperature 101° F., pulse 90), and crepitations could be heard in the right lung. A chest radiograph revealed opacities throughout the right lung, confluent in the upper zone. There were also some less prominent opacities in the left mid-zone. A sputum test revealed the presence of tubercle bacilli.

When first seen, she had already missed a couple of periods and there was the possibility that she might be pregnant. In fact, some of her symptoms of malaise had been attributed to her possible pregnancy. Pelvic examination revealed enlargement of the uterus and a Friedman test on the urine confirmed that she was in fact pregnant, and that the amenorrhœa was not a toxic effect of her tuberculosis.

At this point, it was considered whether or not the ten weeks' pregnancy should be terminated, but it was decided to allow it to continue.

She was admitted to hospital and maintained at complete bed rest.

Chemotherapy was initiated, consisting of streptomycin sulphate 1 gm. intramuscularly daily, together with 15 gm. P.A.S. daily. After 60 gm. of streptomycin had been administered, the drug was continued at the reduced dosage of 1 gm. on alternate days, for a further 30 gm. A total of 90 gm. of streptomycin was therefore given from the tenth to the thirtieth week of pregnancy.

There was a very good response to bed rest and chemotherapy and there were no toxic effects from the latter. There was a rapid subsidence of the toxæmia, the pyrexia and pulse rate soon becoming normal. The general condition of the patient improved greatly. Radiologically there was considerable clearing of the opacities in both lungs, leaving only a smaller circumscribed opacity in the right upper zone, while the left lung now appeared normal. Tubercle bacilli disappeared from the sputum after six weeks.

When the chemotherapy had come to an end, strict bed rest was still maintained throughout the remainder of the pregnancy, and there was further contraction of the lesion in the right upper zone.

Throughout this time, the pregnancy progressed uneventfully. At term, there was a normal confinement, and a male infant was born without any complications. The infant breathed and cried immediately. Birth weight, 8 lb. 11 oz. Head circumference, 13½ inches; length, 20½ inches. The placenta was normal.

The subsequent progress of the mother suffered a temporary setback, in that tubercle bacilli reappeared in the sputum shortly after the confinement, although there was no radiological change. The infant was separated from the mother from birth, and breast feeding was not allowed. Further streptomycin (45 gm.) and P.A.S. were administered, and a right artificial pneumothorax induced. Following the division of a single adhesion she was left with an excellent collapse of her right lung, and the sputum has been repeatedly negative. She was then " up-graded " and her disease has remained quiescent. Mother and infant were reunited at four months, the infant having in the meantime been vaccinated with B.C.G.

The progress of the infant has been normal. At the time of writing, he is aged 1 year and weighs 25 lb. He sits up and crawls normally, plays with objects with dexterity, and there is no unsteadiness in his movements. He behaves generally in a perfectly normal fashion. There has been no nystagmus, and his hearing is normal.

Conclusion

Streptomycin in the dosages usually employed, when administered to a pregnant women, passes through the placenta without producing any toxic effect on the placenta itself. There is some placental barrier to the drug, however, for the concentration of streptomycin in the fœtal blood is usually one half or less than that in the maternal blood, but the fœtal blood level is still usually sufficiently high to produce an antibiotic effect. This may possibly, in some severe infections of the mother, serve as a prophylaxis against congenital tuberculosis. The placental barrier may also be a modifying factor resulting in the innocuous nature of streptomycin to the fœtus, since the fœtus does not appear to be affected adversely by streptomycin administered to the mother, even when given in the first trimester and in prolonged dosage. The infants are then apparently born quite normal and do not show any specific damage to their eighth cranial nerves.

I should like to thank Mr. A. B. Stenhouse, Obstetrician, Kingston Hospital, for his co-operation in the case described.

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REVIEWS OF BOOKS

A Practice of Thoracic Surgery. By A. L. D'ABREU, O.B.E., Ch.M., F.R.C.S. London: Edward Arnold and Co. 1953. Pp. 591. 343 illustrations. Price 80s.

Mr. d'Abreu is to be congratulated on producing a book which covers all aspects of thoracic surgery in a reasonable compass and at the same time keeping it fully up to date. Chest surgery has progressed so rapidly in recent years that even its practitioners find it hard to keep abreast of current developments. The principal merit of the book is that it balances the clinical aspects with the operative procedures and results. The fault of so many other works on this subject has been that they have confined their attention to the technical side. The subject matter is representative of the majority of British thoracic surgeons and shows how a regional speciality has to be based on a sound general surgical training.

The book starts traditionally with anatomy and physiology and proceeds to the surgery of suppuration, where possibly the chapter on empyema is less forceful than the others, though the section on bronchiectasis is concise and clear. Pulmonary tuberculosis is dealt with as fully as this difficult subject can be by generalisations, and when dealing with bronchial carcinoma the follow-up figures from Birmingham provide valuable material in illustration of the text. A considerable amount of space has been given to the surgery of the heart, though out of just on one hundred pages more than a third has been devoted to angio-cardiography and cardiac catheterisation. These sections in themselves are excellent and well illustrated. Modern heart operations are fully mentioned and many of the possibilities for future surgery have been indicated. The section on the œsophagus is most satisfactory and gives an excellent picture of the surgery of this organ at the present time. There is also a section on a number of miscellaneous thoracic abnormalities such as mediastinal tumours, hydatid cysts and injuries. Finally, the work concludes with a section on thoracic abdominal surgery which includes the diaphragm and a number of upper abdominal operations performed through the chest.

The X-ray reproductions are good, as also the photographs of pathological speciments. Perhaps a few more line drawings might have amplified some features of the text, but that is a minor criticism. References at the end of each chapter are not meant to be comprehensive, but are carefully selected and useful. This book is a most admirable addition to current surgical literature.

T. HOLMES SELLORS.

Modern Trends in Diagnostic Radiology. By J. W. MCLAREN. London: Butterworth and Co. 1953. Price 70s.

This book is not a second edition of that published in 1948, but a second volume or series, and the various articles, for the most part by new contributors, are concerned mainly with radiological matters which were omitted then.

For the chest physician there are four articles of considerable interest all conveniently grouped together in the early chapters. The first is that by Schönander, Fredzell and Busch describing recent advances in X-ray apparatus.

The description of an image amplifier for fluoroscopy is an indication of advances in one sphere which may revolutionise our conception of the utility and ease of this method of examination.

The authors are perhaps rather modest in their claims, since the Westinghouse machine is now beyond the experimental stage and available in England. The small size of the screen (some 10 cm.) as yet limits its value in chest work, but a larger screen is apparently being developed.

The next article by Watson on simultaneous multi-section radiography describes an extremely simple piece of apparatus, available to anyone using a tomograph, which enables five layers to be radiographed on five films using only a single swing and exposure. This will be particularly useful in diminishing the dose of X-rays received by a patient in a series of lateral-view tomograms. Two exposures will provide ten layers, 1 cm. apart. The article might perhaps have been enlarged and more detail given, but the author is available at Ilfords at Tavistock Square, to anyone wishing to pursue the matter further.

An article by Pierce on fungous diseases (of the lungs) is wholly admirable and gives a very clear account of the radiological and pathological findings in this difficult group of diseases.

Articles on angiocardiography by Wegelius and Lind, and by Jönsson, will be of great value to those interested in cardiology.

All these sections concerned with intrathoracic diseases are well worth while reading by anyone interested in the radiology of the heart and lungs. The only criticism I would venture to make is that the translation of the Swedish articles would have been better if converted into a clear English than in a literal word-for-word form.

GEORGE SIMON.

Traité de Phthisiologie. By JACQUES STEPHANI, Privat Docent à la Faculté de Médecine de Genève, with the collaboration of Edouard Arnold, Robert Kirsch, François Nicoll, Gilbert de Rham, Jean-Aimé Baumann, P. Naef, André Petit, Gustave Turini. Préface by Professeur Georges Bickel. Paris: Amédée Legrand et Cie. 1953. Price 5,300 francs.

The name of Dr. Jacques Stephani is a guarantee of a well-considered treatise on any aspect of tuberculosis. In this volume he is assisted by a group of collaborators, all of whom are well known for their interest in, and for their experience of, this disease.

The tuberculosis physician who regards himself as a specialist, concerned solely with the state of the lungs, would do well to read the introduction to this book, for emphasis is laid upon the necessity for considering the patient as a whole, and the secondary changes which may occur in other systems of the body as a result of infection in the lungs are considered in some detail.

The first part of the book is devoted to general considerations, and this is followed by a brief description of the methods of physical examination and of the special tests which are essential in the diagnosis of the obscure case. The author agrees with the modern conclusion that, while physical examination of the chest is still an essential part of the examination, and in some cases can provide information otherwise not obtainable, the X-ray examination is far more important. There follows a complete description of all the laboratory and special means of diagnosis in common use, with a clear account of the value of each.

Naturally pulmonary tuberculosis is considered in greatest detail and most extensively, but this section is followed by a somewhat brief description of tuberculous disease as it affects most of the other organs of the body. There is an excellent section on differential diagnosis. The effects of tuberculous infection may cause so many symptoms and so many signs, not only in the respiratory tract but elsewhere in the body, that the differential diagnosis of tuberculosis is in reality the differential diagnosis of most medical conditions. Hence the vital necessity for the tuberculosis physician to be conversant with general medicine. In reading the sections which are concerned with the clinical aspects of tuberculosis one covers much of the ground of medicine. To know tuberculosis is to know medicine. Perhaps the converse should also be true.

The volume concludes with a very complete description of all the methods of treatment in common use, and it is up-to-date in that it includes a useful section on isoniazid. Artificial pneumothorax treatment is described in detail and it is obviously still considered by the author to be a very valuable method of treatment, and one to be employed as a method of choice in suitable cases. The average duration of treatment is considered to be four years, which is a little longer than has been suggested in recent continental writings, but is probably a just estimate of the optimum period. It is a little astonishing to note that oleothorax is still a fairly important method of treatment on the Continent, if we are to judge by the fact that no less than eight pages are devoted to a detailed consideration of this method of treatment. The author is more cautious with regard to pneumoperitoneum, and he appears to regard this as being a less valuable method of treatment than pneumothorax. This is not the view commonly held in this country at the present time, but it is probably an accurate forecast of things to come. The question of resection is dealt with by A. P. Naef, of Lausanne. He obviously writes from the surgical point of view, although he makes it clear that surgery in tuberculosis is simply a part of the general scheme of treatment and no short-cut to a cure.

The book is well printed upon excellent paper. On the whole the X-ray reproductions illustrate clearly the points which they are meant to demonstrate, but some of the films suffer by excessive reduction.

JAMES MAXWELL.

Phthisiologie du Praticien. By M. BARIÉTY and G. BROUET. Paris: Masson et Cie. 1953. Pp. 500. 220 Figures. Price 1,450 francs.

This is the third edition of a book which was first published in 1944 and which has been extensively rewritten to march with the progress of the intervening years. It is written for the general practitioner and the student, and the authors state that it is not for the *spécialistes chevronnés*, although many of these gentlemen might read it with profit even if it is only to refresh their memories. It is illustrated with 220 excellent line drawings and the subject is covered in a comprehensive and eminently readable way. A chapter on radiology deals simply and effectively with the shadows cast by the various structures of the chest wall, the thoracic viscera, and the vascular and glandular tissues. All are illustrated and the sites and shadows of the lung lobes and segments are similarly dealt with. This chapter is of great value in explaining and delineating the many difficulties which arise in the interpretation of films and of the screen image, especially from the point of view of the misinterpretation of the

shadows cast by normal organs and tissues. Diagnosis is treated in a sensible and logical fashion, and treatment is dealt with exhaustively and on common-sense lines. This is particularly so in the matter of the basic routine of treatment and in the use of the antibiotic drugs. It is refreshing to read nowadays of the homely treatment of the patient, his disease, his symptoms and his troubles, and of the useful part which many of the old-fashioned drugs can still play in the complications of this troublesome disease. It is also refreshing to find that pneumothorax still has its adherents who know its possible complications and consequently how to avoid them; but it is surprising in a book of this scope to find 74 pages devoted to this subject. Few practitioners in this country can have facilities for this work and still fewer can have any wish to embark upon it. There are chapters on prophylaxis, on case-finding methods and on the social problems of the disease, and a brief description is given of the French anti-tuberculosis organisation. If the style is often conversational and sometimes repetitive that is a minor fault. The authors take us with them and they have given us a useful book.

CHARLES CAMERON.

Brompton Hospital Reports. Vol. XXI, 1952. Published by the Research Dept. of the Hospital. Aldershot: Gale and Polden, Ltd. Pp. 206. Illustrated. Price 15s.

This latest edition of the Brompton Hospital Reports contains several articles of interest to workers in chest diseases. Among these the reports by Foster-Carter *et al.* and Frank Scadding *et al.* concerning the results of pneumothorax treatment are of particular interest at a time when other newer methods of treatment are proving their efficacy. The conclusions reached in both investigations agree largely and demonstrate that pneumothorax treatment judiciously applied under satisfactory conditions is still an effective measure which is likely to be in use for some time to come. Atelectases, adhesions and small effusions in the course of treatment are shown to be of relatively little significance provided cavity closure is achieved in a reasonable time. The general impression is confirmed that it is the persistently open cavity under an artificial pneumothorax which carries a grave risk of complications and a poor prognosis.

The validity of these findings is beyond question, but whether they are generally applicable is open to doubt. In each investigation the cases under review remained under the control of a single group of physicians throughout and they were managed under uniformly good conditions. It is probable that the same good results would not be obtained among migratory patients who are refilled at several clinics with varying degrees of efficiency, or at the same clinic where there are constant changes of staff.

J. G. Scadding contributes a concise account of the present status of the chemotherapy of pulmonary tuberculosis. This is naturally mainly concerned with streptomycin and P.A.S., their methods of administration and the indications and contra-indications for and against their use. The need for further investigation regarding their possible use in minimal pulmonary tuberculosis without symptoms, tuberculous effusions and primary lesions, is indicated.

The view that pregnancy has no very dramatic effect on the course of pulmonary tuberculosis receives further strong support from the observations

of J. R. Edge on 462 women, among whom there were 54 completed pregnancies in 47 patients.

The difficulties and risks of bronchography in children are well known and any method likely to reduce them is welcome. Bates *et al.* describe a modified technique which they have used without complications and with 80 per cent. satisfactory results in 250 children.

PHILIP ELLMAN.

Hormonal and Neurogenic Cardiovascular Disorders. By WILHELM RAAB, M.D. London: Baillière, Tindall and Cox. 1953. Pp. xxi+722 with 82 illus. Price 114s.

This is a very important book written by a professor of experimental medicine who is also a leading clinician and head of the Cardiovascular Research Unit at the University of Vermont.

The first section appropriately commences with the Adreno-Sympathogenic Catechoalamines (Epinephrine and Nor-Epinephrine), because it is these known pressor substances and other unknown ones which may play an important part in hypertension. The adreno-cortical hormones, both mineral corticoids and glucocorticoids, which are probably of equal if not greater importance in considering the fundamentals of cardiovascular problems, are ably considered in the next section. All hormones, however, receive attention from the experimental and chemical angle.

Although the next section is labelled " Cardiovascular Features in Endocrine Syndromes," it is, in fact, a comprehensive and readable description of major endocrinopathies. One is, however, immediately faced with the fundamental difficulties of an unravelled complex situation, because hypertension is a manifestation of so many endocrine hyperfunctions—*e.g.*, adrenal medullary tumours, adrenal cortical tumours, thyrotoxicosis, myxœdema, acromegaly, the climacteric, hyperparathyroidism with renal involvement and adiposity with some stigmata of endocrine dysfunction.

The third section is an attempt to estimate how far endocrine factors are causal or aggravating in cardio-vascular syndromes, in the light of the knowledge that any or all of the endocrine glands may be involved in the answer. It is primarily a cardiovascular section and of very great value from this aspect; but the absence of direct, convincing implications of the endocrine glands in the majority of cardiovascular disorders, combined with the lack of other evidence of endocrinopathies, renders exploration in this field inevitably incomplete and disappointing. For what they are worth, the results of my own observations, clinical and experimental—the latter in co-operation with laboratory colleagues—lead me to believe that, apart from the well-known endocrinopathies, there is an endocrine constitution, and that when this constitution involves a pituitary-adrenal hyperfunction there is a basis for cardiovascular disease in a number of patients who are not suffering from obvious endocrinopathies.

The integrity of the writing and observations in this book renders it a work of the very greatest value, and it constitutes a classical work in this field which will probably be the forerunner of much creative and exploratory investigations. It is a book which every physician interested in the basic sciences of his field will want to read and keep.

S. LEONARD SIMPSON.

Die Wirbelsäulentuberkulose und ihre Differentialdiagnose (Tuberculosis of the Spine and its Differential Diagnosis). By J. E. W. Brocher. Stuttgart: Georg Thieme Verlag. 272 pages, List of References, Index. March, 1953. Price D.M. 78.

The title is not quite adequate, as only a third of the book deals with tuberculosis of the spine, while two-thirds are concerned with other spinal conditions, some of them totally unrelated to tuberculosis even from the viewpoint of differential diagnosis.

If the author's primary aim is to offer a monograph on tuberculosis of the spine, the problems of prognosis and treatment appear as rather neglected. Up-to-date information about the value of antibiotics is particularly scanty. Ankylosing spondylitis is fully described in 33 pages, but Romanus' studies have escaped the author and the importance of genito-urinary infection in this condition is not discussed. Scheurmann's disease has its full share of 27 pages, but I do not think Scheurmann will agree with the interpretation given to all the radiographs.

Other conditions, such as osteochondrosis, vertebra plana, congenital deformities and tumours causing decalcification, are well illustrated. Radiographs showing spinal changes due to intraspinal tumours are not included.

This is a comprehensive book presented in an attractive form. Its most valuable part is the wealth of illustrations, particularly the extremely well reproduced radiographs.

J. Agerholm.

BOOKS RECEIVED

The following books have been received and reviews of some of them will appear in subsequent issues:

Sectional Radiography of the Chest. By Irving J. Kane, M.D. New York: Springer Publishing Co. Inc. 1953. Price £3.

Aspects of the Psychology of the Tuberculous. By Gordon F. Derner. 1953. New York: Harper and Bros. Pp. 119. Price 25s.

Clinical Cardiology. Edited by Franklin C. Massey, with 33 Collaborators. 1953. London: Baillière, Tindall and Cox. Pp. xiv + 1100. 250 Illus.

Respiratory Function. By Richard D. Tonkin. Physical Medicine Series, Vol. II. 1953. London: Actinic Press Ltd. Pp. 64. 11 Illus. Price 3s. 6d.

Tuberculosis in B.C.G. Vaccinated and Non-Vaccinated Young Adults. A comparative Prognostic Study by Gunnar Dahlstrom. Copenhagen: Ejnar Munksgaard. 1953. Pp. 138+iv. Price 17s.

Pneumoconiosis Abstracts, Vol. I. Pitman. Pp. 1-347 + i-xi, with illus. 1953. Price 63s.

Tuberculosis Index and Abstracts of Current Literature Quarterly. September 1953. Vol. 8, No. 2. N.A.P.T. Price 25s., per year $5.

Journal of the Royal Sanitary Institute, Vol. LXXIII, No. 6. Pp. 1-790 + i-xxvii, with illus. Price 4s.

Health Horizon. Autumn, 1953. N.A.P.T. Pp. 56, with illustrations. Quarterly, Price 2s. 6d.

Report on the Health of Birmingham, 1952: with a contribution on Tuberculosis by Dr. J. E. Geddes. Pp. 310.

Funktionelle Atmungstherapie. By Dr. J. Parow. 1953. Pp. xii+126. 29 illus. Stuttgart: George Thieme Verlag. D.M.12.90.

Strahlenschutz und sonstiger Arbeitsschutz bei der medizinischen Anwendung von Rontgenstrahlen. By Dr. W. Ernst. 1953. Pp. vi+97. Illus. 22. Stuttgart: George Thieme Verlag. D.M.7.80.

I Bronchi di Drenaggio delle Caverne Tubercolari del Polmone (Studio Anatomo-Istologico). By Antonio Blasi. Preface by V. Monaldi and P. Verga. 1953. Naples: Editrice Archivio di Tisiologia. Pp. 1-319. Figs. 179. (No price indicated.)

REPORTS

B.C.G. VACCINATION OF SCHOOL CHILDREN

Local health authorities in England and Wales have been informed by the Minister of Health, Mr. Iain Macleod, that he is prepared to approve schemes for giving B.C.G. vaccination to children before they leave school.

Since 1949, when official permission for its use in this country was first given, B.C.G. vaccination has been offered to nurses and medical staff in hospitals and (at the discretion of chest physicians) to the close home contacts of tuberculosis cases.

It will be for local health authorities themselves to decide whether they wish to undertake the vaccination of school children. Where it is done it will be offered, in co-operation with the education authority, to children of thirteen years of age. The consent of parents will always be first obtained.

Discussing the value of B.C.G. vaccination, a medical memorandum which Medical Officers of Health have received from the Ministry mentions that the Medical Research Council have already undertaken extensive controlled trials among school leavers. The results, it is hoped, will assess the protective value of B.C.G. as a means of mass immunisation of persons in ordinary average conditions of life. The results of these trials will, however, not be known until the subsequent history of the children concerned has been followed for about three years.

N.A.P.T.

Leadership against Tuberculosis

The Annual Report for 1952-53 has been received. It shows some of the magnificent voluntary work that continues to be done by the Association and deals briefly with such problems as new drugs and their consequences, case-finding, X-ray methods, B.C.G. vaccination, research problems, care work, tuberculosis in the Commonwealth, international union against tuberculosis, etc.

The report makes interesting reading, and shows how very much the Association is taking a real lead against tuberculosis; its interests are confined not merely to treatment but in equal measure to the prevention of the disease.

MINISTRY OF LABOUR AND NATIONAL SERVICE

Report on Resettlement of Disabled Persons During the Year 1952

Extracted from the Annual Report of the Ministry of Labour and National Service for 1952. Cmd. 8893

Concerning tuberculosis, the report makes the following observations:

Many employable tuberculous persons are able to work under ordinary industrial conditions so long as care is exercised in choosing the right kind of work for them and in ensuring that there are effective safeguards in such matters as personal hygiene. For those who needed sheltered conditions employment was provided in the speci l Remploy factories and also in Village Settlements run by voluntary undertakings. In these settlements the Ministry continued to give financial assistance towards the training of tuberculous persons from the stage at which they could work only a few hours daily until they could work full hours and either become employees of the Settlement or find suitable work outside.

The responsibilities of Local Authorities in relation to the provision of workshop employment for tuberculous persons were reviewed, and it was decided that such facilities should continue to be dealt with under Section 28 of the National Health Service Act (or Section 27 of the National Health Service (Scotland) Act, 1947). This meant that this Ministry would continue to be responsible only for training ex-tuberculous patients, including the payment of maintenance allowances to the trainees and training fees and other incidental expenses to the establishments, leaving local authorities to provide employment facilities under the terms of the National Health Service Acts.

ROYAL VICTORIA HOSPITAL TUBERCULOSIS TRUST: ANNUAL REPORT 1952-1953.

The Committee of Management make certain important observations on the tuberculosis position in Scotland.

Recent Mortality Trends in Tuberculosis

The statistical returns for the year 1952 have continued in one respect the favourable progress which the Committee have commented on since 1948. The death-rate from respiratory tuberculosis in 1952 was 27 per 100,000 of the population and from non-respiratory tuberculosis the corresponding figure was 5, giving a mortality from all forms of the disease of 32 per 100,000. This is the lowest figure on record, and the death-rate from tuberculosis (all forms) has diminished by no less than 51 per cent. in the last four years. It may now be stated that at last the stage has been reached which would have been attained by this time had the pre-war trends in mortality continued without interruption.

Despite this generally favourable statement as to progress, expert investigators know well that there is a much more sinister side to the problem of respiratory tuberculosis in Scotland. We have not shared in the general improvement to the extent that many countries have done, as, for example, England and Wales, Belgium, Holland, and all the Scandinavian countries except Finland. In particular, we have not shared the favourable progress made in young age-groups in these countries. In Scotland the death-rate from tuberculosis of the lungs in young men aged 15-24 is half as much again as it

is in England, whilst the mortality among young women in this age-group is actually twice as high. It should be remembered that in Scotland, whilst the mortality from tuberculosis at all ages before the war was slowly dropping, the mortality among young people had already begun to rise. In young men the death-rate began to rise about 1938 and in young women about 1934. The serious effects observed from 1940 onwards among young people were therefore not wholly due to the war, though they were greatly exaggerated by it. While effects of the war, however, are now over as regards tuberculosis as a whole, the position in the age-groups 15-24 is still serious. The Committee noted a slight improvement even in this vulnerable group in their last report (1950) and are glad to observe that the improvement has continued this year (1951), though much leeway still remains to be made up before the Scottish figures can compare with those for England and Wales.

It cannot be reiterated too often that the death-rate does not tell the whole story. The tuberculosis position in relation to new cases is still very disquieting.

New Notifications

The provisional figure for notifications of respiratory tuberculosis in 1952 was 7,259, compared with 7,776 for the previous year and 8,653 in the " crisis year " of 1949. The progress recorded is equal to a drop of 15 per cent. in the last three years. Welcome as this improvement may be, the annual number of notifications is still about 64 per cent. higher than the lowest number recorded in 1936, and of the total number of new notifications as many as half are among young people. The actual increase is almost certainly greater than these figures indicate. In 1936 only 74 per cent. of notifications were subsequently confirmed (3,544). In 1951, 97 per cent. were confirmed (7,555). It may be justifiably assumed, therefore, despite the improvement since 1949, that the increase in fresh cases which has taken place between 1936 and 1951 is actually about 108 per cent.

Whilst the Committee welcomes the drop in notifications in 1952, the figure should be accepted with caution. The provisional returns for the last quarter of 1952 and the first quarter of 1953 both show an increase over the corresponding quarter of the previous year. It may be that a retrogressive tendency has again begun.

The Tuberculosis Register

Since the annual number of confirmed notifications (despite recent improvement) is still twice as high as it was, and since the mortality rate continues to fall, it is not surprising that the number of survivors is increasing. Thus the number of known sufferers from respiratory tuberculosis on the tuberculosis register increases year by year. In 1930 there were 22,193 known sufferers. The figure fell slowly in pre-war years, reaching a minimum in 1938 of 17,465, but rose during the war and reached 24,587 in 1945. Thereafter the increase has gone steadily on, and in 1951 there were no less than 37,110 persons on the register, or more than double the figure for 1938. Further analysis of the figures shows that once again the emphasis of this increase is in young people. Whilst, in 1930, 48 per cent. of persons on the register were aged 15-34, in 1951 this figure was 60 per cent.

The Committee, therefore, wish to state again with all the emphasis they can command that the publicising each year of an overall death-rate figure which is stated to be yet another low record for tuberculosis does a public

disservice, since it creates in the mind of the people the erroneous impression that tuberculosis is "on the run." The information that the annual number of confirmed new cases and the total number of known sufferers are over twice what they were before the war receives less publicity. The fact that in Scotland the statistics are worse than in most countries of Europe from which reliable figures are available is little appreciated.

Future Requirements

Future policy must rapidly develop in many ways, the three most important being—

(1) Expansion of the Tuberculosis Hospital Service.
(2) Case finding measures.
(3) B.C.G. vaccination, especially of the school leavers.

The need for the protection of the adolescent and young adult is emphasised.

PAPWORTH VILLAGE SETTLEMENT

We have received the Annual Report of the Papworth Village Settlement for the year 1952. The Report comprises that of the Committee of Management, the Report of the Medical Director, an Industrial Report for 1952 by the Managing Director and a note of the social life of the Settlement during that year.

An interesting follow-up of trainees was instituted to assess the results of the Ministry of Labour's training scheme. Information gathered about the 346 who entered the scheme between April 1948 and March 1952 has been prepared by Dr. Owen Clarke, which makes interesting reading and clearly supports, states the Medical Director, the view long held at Papworth that the tuberculous, whether trainees or colonists, are reliable workers, especially once they have attained a position of self-support. Dr. Trail also adds that it demonstrates the extreme value to all patients of the Ministry's generous scheme and the fact that the vast majority of patients recognise to the full the responsibilities that accompany its privileges.

PRESTON HALL HOSPITAL

Second Report of the Management Committee: April 1, 1951, to March 31, 1953

Among the observations made in this Report, it is stated that, in spite of continued staffing difficulties, it has been possible to open more beds, and the capacity of the hospital is now 330 beds, of which 75 are reserved for thoracic surgical cases and 105 for ambulant cases. In addition there are the associated beds in the Rehabilitation Unit controlled by the British Legion. These number some 135, but are available for use by hospital patients if required, under the "Joint User" arrangement between the British Legion and the Regional Hospital Board. The opening of the extra hospital beds has been made possible by an improved recruitment of nursing staff and particularly by the arrangement made with King's College Hospital for the secondment of ten student

nurses every quarter. Moreover, as a result of this arrangement, several King's College nurses, who have since qualified, have applied to return in order to take their British Tuberculosis Association Certificate in tuberculosis nursing.

ANNUAL REPORT OF THE COUNTY MEDICAL OFFICER OF HEALTH FOR THE YEAR 1952, DERBYSHIRE COUNTY COUNCIL

In the course of this report a number of interesting observations referable to the tuberculosis scheme are made.

It is satisfactory to note that the arrangements for prevention, care and after-care are closely co-ordinated with the diagnostic and treatment services, and in this connection Dr. Kingston, one of the Consultant Chest Physicians, points out that a regular exchange of information between the Chest Clinics in the area and the County Medical Officer works smoothly and effectively. Chest Physicians are informed by the County Medical Officer of all new notifications of tuberculosis, and Health Visitors' reports on home conditions of tuberculous patients are also sent to the Chest Physician concerned, as also are particulars of deaths from tuberculosis, patients' changes of address, etc., which become known to the County Medical Officer. Weekly returns from Chest Clinics inform the County Medical Officer of new cases seen, removals from Clinic Registers (recovered cases, transfers to other areas, etc.) and changes of address. The County Medical Officer is also informed of admissions to and discharges from sanatoria.

Great attention is paid to the preventive sphere, and it is interesting to read, among other observations:

"As in the past, it is in the field of prevention that the biggest gains are likely to be made in proportion to the energy expended, and we feel that it is in this direction that our efforts are least employed. Mass Miniature Radiography touches only the fringe of the population, and although a lot of new treatable cases are found by this means, employed in its present form it is unlikely that it exerts even a small effect on the prevention of disease. We are of the opinion that it can best be used in a concentrated attack on the 'black spots' of the country in an endeavour to X-ray 100 per cent. of the population in these areas."

The Annual Report on the Health and Sanitary Conditions of the County Council of Dumfries for the Year 1952

Dr. Drainer reports that there is a steady fall in the death-rate from tuberculosis of all forms in Dumfriesshire, as shown by the following figures:

1901–10	2·76
1911–20	1·38
1921–30	0·99
1931–40	0·59
1941–50	0·47
1951	0·17
1952	0·10

It is known that 402 persons in Dumfriesshire suffer from tuberculosis—327 respiratory cases and 75 non-respiratory cases. Six persons died from

tuberculosis during the year—in each case the disease was of the respiratory type. There were no deaths from the non-respiratory forms of the disease.

The tuberculosis death rate of 0·10 per 1,000 of the population is by far the lowest ever recorded in Dumfriesshire. Much of the credit for this favourable picture is the proven efficacy of the antibiotic drugs in the treatment of the disease.

THE NATIONAL B.C.G. COMMITTEE
ST. ULTAN'S HOSPITAL, DUBLIN

We have received the report for the year ending December 31, 1952. In summary, it states that the National B.C.G. Committee was set up in July 1949 by the Minister of Health, for the purpose of directing and being responsible for the expansion of B.C.G. vaccination in Ireland. The work of the Committee is on a voluntary basis, written permission being obtained before vaccinations are carried out. The services of the Committee's vaccinators are available to medical officers of health, dispensary and private practitioners, doctors to factories, schools, institutions, etc.

In this, the third Report of the Committee, the work carried out during 1952 is examined. During the period January to December inclusive, 170,698 preliminary tuberculin tests were done; 65,401 B.C.G. vaccinations; 365 re-vaccinations and 49,262 post-vaccinal tuberculin tests. Those vaccinated have been analysed according to area, age-group, sex, contact history and institution. A post-vaccinal conversion rate of 98·41 per cent. has been obtained.

The work of the Committee from its initiation in July 1949 has already been published in Reports covering the periods July 1949 to December 1950 and January to December 1951. The total figures for the three-and-a-half year period ending December 1952 are: 279,737 preliminary tuberculin tests; 115,992 B.C.G. vaccinations; 957 re-vaccinations and 87,281 post-vaccinal tuberculin tests.

NORTHERN IRELAND TUBERCULOSIS AUTHORITY:
REPORT FOR YEAR ENDED DECEMBER 31, 1952

In the 7th Annual Report of the Northern Ireland Tuberculosis Authority, reference is made to the importance of education and propaganda.

"The work of the special department established to deal with education and propaganda is still expanding. Numerous publications were produced in the course of the year for distribution to the general public and/or to special groups within the community.

The use of display stands at agricultural shows remains a feature of the publicity campaign, and unquestionably the members of the public continue to evince an increasing interest in the propaganda material exhibited.

Various other ways and means of educating patients and members of the general public are being pursued. Amongst other things, it is hoped shortly to issue a booklet for the guidance of patients, whilst plans are being made to conduct an educational campaign through the medium of newspapers circulating throughout Northern Ireland.

The need for widespread publicity is keenly recognised by the Authority, whose intention it is to use propaganda on a comprehensive scale in the belief that a community which knows the facts about tuberculosis is one which will wipe out the disease in the shortest space of time."

WORLD HEALTH ORGANISATION

Recommendations of W.H.O. Conference in London

"Vital and health statistics are essential tools for the formulation and administration of public health programmes" and should accordingly be developed in every country.

This basic principle was reaffirmed at the first International Conference of National Committees on Vital and Health Statistics, convened under the auspices of the World Health Organisation in close collaboration with the United Nations.

The conference took place in London, from October 12 to 17, at the General Register Office, Somerset House, and was attended by twenty-eight delegations from W.H.O. Member and Associate Member States. Representatives of the International Labour Office and of the International Statistical Institute were also present.

Sir Walter Russell Brain, President of the Royal College of Physicians, was appointed honorary president of the conference by Her Majesty's Government. Professor A. Bradford Hill, of the London School of Hygiene and Tropical Medicine, was unanimously elected chairman of the conference, and Professor Stefano Somogyi, of the Central Statistical Institute, Rome, and Major Dr. H. Vejjavisit, Director-General of the Department of Medical Service, Thailand, vice-chairmen.

The conference noted with satisfaction the important activity of the National Committees on Vital and Health Statistics created in thirty countries, one of whose objectives is "to relate the activities and functions of diverse agencies or organisations that produce statistics, so that they work as a co-ordinated whole, avoiding both wasteful overlapping of effort and important gaps in essential parts of statistical data."

CANADIAN TUBERCULOSIS ASSOCIATION

A dramatic reduction in the number of deaths from tuberculosis in Canada during 1952 has been reported by the Dominion Bureau of Statistics in submitting its most recent figures. Deaths fell from 3,422 in 1951 to 2,457 in 1952 and the rate is down from 24·5 to 17·1, a drop of seven points, or more than 30 per cent. This is the largest reduction to be reported in any one year, and would seem to indicate that the steady downward trend in tuberculosis mortality is being accelerated.

When the overall figures are studied with respect to age, a further interesting fact emerges—namely, the large number of deaths occurring over age thirty. Out of a total of 2,457 for all of Canada, 1,690 were among people past thirty. It can no longer be said that tuberculosis is a young person's disease.

THE NATIONAL ASSOCIATION FOR THE PREVENTION OF TUBERCULOSIS IN AUSTRALIA

THE Annual Meeting of the Council was held in Hobart, Tasmania, on October 2, 1952.

1. *Proposed Pacific and East Asian Tuberculosis Conference.* Correspondence with W.H.O. and the Commonwealth Government indicated that there was a strong possibility that this would be held in Sydney in August 1955.
2. It was recommended that the Laennec Society should organise the scientific programme of this Conference, in collaboration with W.H.O.
3. It was decided to contribute £100 as a membership donation to the funds of International Union against Tuberculosis for the current year.
4. Suggestions were made for increasing the educational activities of N.A.P.T.A. in the fields of broadcasting, films, pamphlets and booklets.

The third Annual General Meeting of N.A.P.T.A. was held after the Council Meeting, when the guest speaker, Dr. Turnbull, Tasmanian Minister for Health, told members of the progress made in that State in tuberculosis control, with special reference to compulsory X-ray examinations, which had proved most successful.

THE AUSTRALIAN LAENNEC SOCIETY

THE Australian Laennec Society held a scientific meeting on the evening of October 1, when the President elect, Dr. Bruce White, entertained some forty guests, medical and lay, at the Tasmanian Sanatorium with a brief account of the value of the Mantoux tuberculin test, and of the difficulties attached to X-ray diagnosis of chest diseases. He also showed some " talkie " films illustrating the educational value of this type of propaganda.

The annual general meeting of the Society was held on the morning of October 3. It was announced that Western Australia had now established a branch, and the other States hoped to follow suit in the near future. The new office bearers elected were:

President: Dr. Bruce White, N.S.W.
Past President: Dr. Cotter Harvey, N.S.W.
President Elect: Dr. J. Thorburn, Western Australia.
Hon. Sec.-Treasurer: Dr. Alan King, Western Australia.

NOTES AND NOTICES

TUBERCULOSIS EDUCATIONAL INSTITUTE

PROGRAMME OF COURSES—1954

DENMARK AND NORWAY

May 9 to 23, 1954

A PARTY will leave London on May 9 to visit Denmark and Norway, spending approximately a week in each country. It is estimated that the inclusive cost (based on third-class travel) will not exceed £50. The programme will include lectures, visits to sanatoria and clinics and sight-seeing. Early registration is essential in order that party-travel concessions may be arranged.

BRISTOL

"*Case Finding and its Implications*"

September 27 to October 1

"*The Psychological Approach to Tuberculosis*"

(for Nurses and Social Workers)

September 28 to October 1

CLINICAL COURSES

SULLY HOSPITAL, GLAMORGAN

January 20, 21 and 22

The following subjects of special interest will be included in the programme: The use of Posture in the treatment of Pulmonary Tuberculosis; Some Aspects of Primary Tuberculosis; Respiratory Function Assessment; Surgical Methods in the treatment of Pulmonary Tuberculosis; The Diagnosis of Pneumoconiosis; Bacteriology in Relation to Chemotherapy; Pulmonary Heart Disease; Asthma; Pathological Aspects of Pulmonary Tuberculosis; Non-tuberculous Chest Diseases.

KING GEORGE V HOSPITAL FOR DISEASES OF THE CHEST, GODALMING

March 3, 4 and 5; September 29, 30, and October 1

RED CROSS SANATORIA OF SCOTLAND (TOR-NA-DEE AND GLEN O' DEE)

April 7, 8 and 9

CHESHIRE JOINT SANATORIUM, MARKET DRAYTON, SALOP

April 28, 29 and 30; October 27, 28 and 29

Further information may be obtained from the Secretary, Tuberculosis Educational Institute, Tavistock House North, Tavistock Square, London, W.C.1.

N.A.P.T. HUNTER SCHOLARSHIPS 1954

THE National Association for the Prevention of Tuberculosis announces four scholarships for doctors and medical students, to be known as the Hunter Scholarships.

Two are postgraduate scholarships of £350 each, one to enable a graduate of a British Medical School, resident in the United Kingdom, to spend some six months abroad studying the control of tuberculosis, the other to enable a graduate from any medical school in the British Commonwealth outside the United Kingdom to study tuberculosis in Great Britain.

Two scholarships of £150 each are offered to medical students of either sex who have been seriously handicapped through tuberculosis, to enable them to complete their training at any Medical School in England or Wales.

These four new awards are in addition to the N.A.P.T. Canadian Exchange Scholarships, valued at £350 each.

The eight N.A.P.T. scholarships awarded each year to doctors, nurses and sanitary inspectors, whose names have been submitted through the Colonial Office by various Colonial Governments, are now open to candidates from all parts of the British Commonwealth.

Enquiries concerning the above scholarships, and other awards made by the N.A.P.T., should be sent to: N.A.P.T., Tavistock House North, London, W.C.1, before March 31, 1954.

THE THORACIC SOCIETY

THE Spring Meeting will be held at the Royal College of Surgeons, Lincoln's Inn Fields, W.C.2, on Friday and Saturday, March 5 and 6, 1954. The Summer Meeting will be held in Manchester on Friday and Saturday, July 16 and 17, 1954.

WORLD HEALTH ORGANISATION

WE have received a film strip, frame size 24 mm. by 18 mm., the title: "The Fight Against Tuberculosis."

The introductory part of this strip explains why people who live under bad conditions are especially exposed to the danger of tuberculosis, and gives a brief explanation of the disease.

The second part of the strip gives an account of the work being done in the prevention and treatment of tuberculosis by the doctors and nurses of the Tuberculosis Centre in Istanbul, which was set up by the Turkish Government in 1950, in co-operation with the World Health Organisation.

NEW RESEARCH INTO TUBERCULOSIS

HUNGARY is setting up a National Tuberculosis Institute in the former Manfred Weiss Hospital for Lung Diseases in Budapest.

The Institute will extend the work of preventing and curing tuberculosis to cover the part played by the central nervous system in the progress of tuberculosis.

Close by the Institute is the 800-bed Koranyi Sanatorium for Lung Diseases, which is to be extended into the largest and most modern curative institute for lung diseases in the country.